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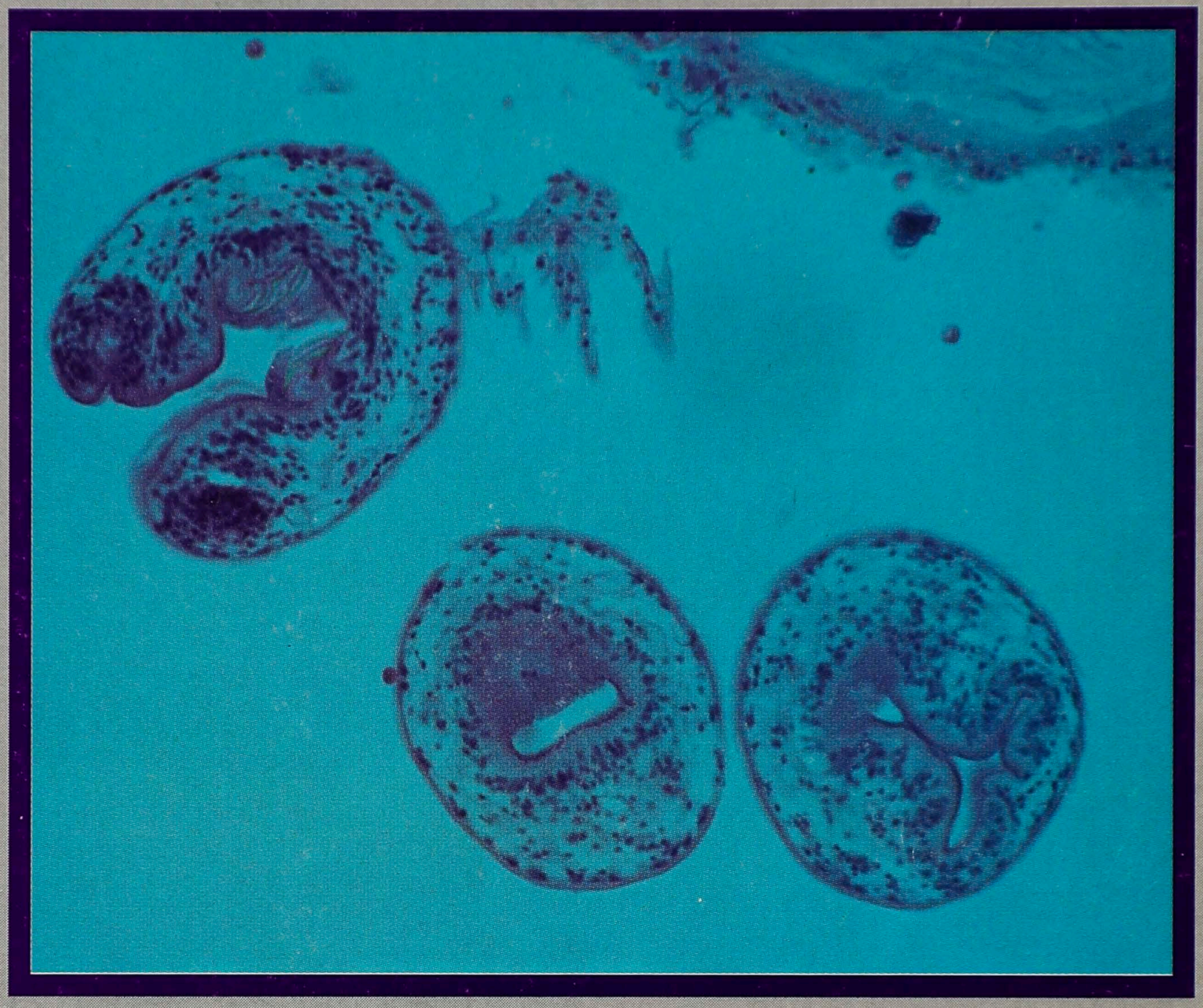
Abdominal Aortic Aneurysms in Canada

Hydatid Lung Disease in Northwest Canada

Desmopressin in Cardiac Surgery



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[1]Eubanks, S et al. Reduction Of HIV Transmission During Laparoscopic Procedures. Surgical Laparoscopy And Endoscopy Vol 3. No. 1, 1993.
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Echinococcal protoscolices from hydatid cyst of lung. (See article on pages 20 to 22.)

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Canadian Journal of Surgery
1867 Alta Vista Dr.
Ottawa, ON K1G 3Y6
Phone (613) 731-9331
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On trouvera des renseignements détaillés à l'intention des contributeurs, en anglais et en français, aux pages 76 et 77 de la livraison de février 1994.

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Defining Abdominal Aortic Aneurysms

Alan M. Graham, MD, FRCSC, FACS

In this issue (pages 29 to 32), Moher, Cole and Hill from Ottawa attempt to define abdominal aortic aneurysms (AAAs) and to look at the management of small AAAs by Canadian vascular surgeons. Their paper is the result of a mail survey to members of the Canadian Society for Vascular Surgery. The authors strive to tell their readers how Canadian vascular surgeons define AAAs, how they measure them and when they treat them electively.

In 1966, Szilagyi and colleagues[1] stated that AAAs required operative management if they were 6 cm in diameter or larger. Data by Bernstein and associates[2] in 1976, however, showed that aneurysms 5 cm in diameter do rupture. As a result, the most common aneurysm size at which surgeons carry out elective repair of an AAA in the good-risk patient is 5 cm.

Moher, Cole and Hill found that of 149 respondents who fully completed their survey questionnaire, the majority operated electively on aneurysms that were 4.5 to 5.4 cm in diameter. However, some operated on aneurysms as small as 4.0 cm in diameter and others waited until the aneurysm was larger than 5.4 cm. All Canadian surgeons who responded to the questionnaire used ultrasonography as the method of sizing AAAs, a finding that is likely related to the cost-effectiveness and accuracy of this method. Only 17% of surgeons used computed tomography, probably because of the lack of its availability in some centres and because of a conscious effort by the surgeons to minimize cost.

The main purpose of the paper, as stated in its title, is to find out how Canadian vascular surgeons define AAAs. The authors offer four definitions of an aortic aneurysm,[3–6] including the most popular one by an International Society for Cardiovascular Surgery (ISCVS)/Society for Vascular Surgery (SVS) subspecialty group.[6] Not surprisingly, there was a wide variety of responses, with all four definitions gaining some support. My questions to the authors are, What do these data tell us? and Do they say anything other than that there is no strict definition of an AAA, let alone strict indications for surgery?

This study would have been much stronger had it followed criteria that have been set by multicentre randomized studies such as the North American Symptomatic Carotid Endarterectomy Trial (NASCET)[7] for carotid artery disease. That is, the study should have defined what an aneurysm was, so that data could be accumulated throughout the country on the basis of that definition. The success of the NASCET was because the definition of stenosis was made by strict criteria, allowing surgeons to enter these criteria accurately.

I find no fault with the Ottawa group trying to find out how Canadian surgeons define and manage small AAAs, but I do have difficulty finding the link between their statistics and their conclusions. Their data confirm what we already suspected: that some surgeons operate electively on "larger" aneurysms and some on "smaller" aneurysms, with the majority "in between." What we need in Canada is for the Ottawa group to define what an aneurysm is and to base a scientific protocol on this definition to provide meaningful prospective results that may have an impact on how Canadian vascular surgeons treat AAAs.

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From the Department of Surgery, Robert Wood Johnson University Hospital, University of Medicine and Dentistry of New Jersey, New Brunswick, NJ

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Reprint requests to: Dr. Alan M. Graham, Department of Surgery, UMDNJ, Robert Johnson Medical School, 1 Robert Wood Johnson Place, New Brunswick, NJ 08903-0019, USA

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The Surgeon's Ego: Moulding the Demon to the Needs of the '90s

Nelson S. Mitchell, MD, FRCSC;* Marilyn Kaplow, BScPT, MSc(A);† Charles McDougall, BA, MHA‡

Surgery has no parallel in human experience. The violent intrusion into the body of an accepting fellow-being for the ultimate benefit of the violated is a powerful experience. After continued repetition, some surgeons become muddled as to which party has the rights and which the privilege. The surgeon who traditionally had a large voice, made large incisions and had great clout has been replaced by one who had less voice, makes tiny incisions (videoendoscopic procedures) and has diminishing clout. Modern hospitals are preoccupied with providing better and smarter management. Managers are selected for these abilities. At the same time, hospital administrators recognize that surgeons are integral to the management process.

Nevertheless, the skills necessary to manage effectively are acquired not inherited. Most surgeons do not possess these because the acquisition of such skills was not part of basic or postgraduate surgical training. Moreover, in the past there was no need for surgeons to learn these skills. Now it is difficult to convince that powerful ego, which has made so many vital surgical decisions, that others may know better how to manage the operating room, the office, the clinic or for that matter any aspect of the hospital. The surgeon has been conditioned to believe that he is the best judge of what is best for the patient and, by extension, every process around that patient. Lang, in his 1991 book *Medical Staff Peer Process*, stated, "The physician has the unique responsibility for determining the special patient needs and managing the system so they are met."[1] To this we would now add the maxim that patient needs and the management of the system must be aligned with the social and fiscal realities of today.

The Surgeon's Ego is easily confused as to its command/management jurisdiction. In the primary

*Senior surgeon, Royal Victoria Hospital, Montreal, Que.

†Manager, Department of Utilization Management, Royal Victoria Hospital

‡Executive vice-president, Royal Victoria Hospital

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Correspondence to: Dr. Nelson S. Mitchell, Department of Surgery, Royal Victoria Hospital, 687 Pine Ave. W, Montreal, QC H3A 1A1

workplace, the operating room, is it the operation, the operating room staff, even the anesthetic that the surgeon should control? The Surgeon's Ego has been nurtured in an environment where instruments were provided, more often on command than by request, by persons of different training and gender. The Surgeon's Ego does not always recognize when it has been abusive, exploitive or patronizing. It does not adapt easily to the new ethos of the '90s, which heralds teamwork, participative decision making and decentralized responsibility. Yet adapt the Surgeon's Ego must, if the surgeon is going to succeed in this environment. Managers negotiate, delegate, lead and above all respect the managed. The surgeon who would be a manager must possess these skills.

Fortunately a new breed of surgeon is developing slowly, albeit in small numbers. Gender equality and opportunity, together with the diverse work experiences and training of the present generation make the younger surgeon more accepting of the skills and knowledge of others. Concepts such as total quality management (TQM), resource utilization review (RUR) and utilization management (UM) are becoming as familiar to the surgeon by their abbreviations as is PTT.

As hospitals continue to develop departments of UM and place greater reliance on data derived from increasingly accurate management information systems (MIS), surgeons must muster their considerable talents to "join the game," to ensure that patients' needs are met without wasting resources. Surgeons must resist the temptation to dismiss such progress under the aphorism, "It will be unsafe for patients." This is not always true, and when it is, it is the surgeon's job to make it safe. We would not have Medicare today if the dire prophecies of doctors had been heeded.

The surgeon is well trained in measuring clinical effectiveness. The statistical manipulations required to validate data and the conclusions springing therefrom are well known to the surgeon. The ability to analyse a problem decisively are part of the surgeon's training and skill. Now the surgeon must commit these skills to cost-effectiveness, efficiency and management decisions taken in concert with the rest of the hospital community. The surgeon's peer group has suddenly enlarged, and the newcomers should be welcomed, for they have a lot to teach that can apply to other aspects of the surgeon's personal and professional life. Surgeons will find the newcomers' approaches to managing the hospital refreshingly different if only they will listen, learn and then contribute their own abundant talent. Patient advocacy depends on the surgeon. It is a traditional role, which must be used now with the utmost honesty, for any suspicion that the surgeon's advocacy is tainted with self-interest will derail the process and interfere with its collective success.

The advantages for the institution and the patient are clear. What is in it for surgeons? The knowledge acquired from participating in the review and the decisions on the allocation of all surgical resources should enable surgeons to be more comfortable with the portion allotted to their own area of interest. This should result in increased surgical volume per dollar spent. Rather than seeing beds as an indicator of authority and responsibility, the surgeons' attention will be focused on patients and the volume of treatment carried out in the operating room or elsewhere. By so doing, surgeons will maintain or perhaps increase their revenue level.

He or she who takes charge of the Surgeon's Ego and undertakes this venture with optimistic enthusiasm will find a new dimension of personal satisfaction. The well-being that derives from serving patients, the institution and ultimately himself or herself will make the effort more than just worthwhile.

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The National Breast Screening Study: a Postscript OR Back to Basics

To the editors. I view the National Breast Screening Study (NBSS) 1 year after its publication[1,2] from the perspective of retirement. In that study, women aged 40 to 49 years were randomized to undergo annual mammography and clinical breast examination (CBE) or usual care after an initial CBE. Women aged 50 to 59 years were randomized to receive an annual mammography and CBE or annual CBE only. The 50- to 59-year age trial was designed to study the additional contribution of annual mammographic screening to screening by CBE alone. No reduction in the death rate from breast cancer was noted in either age group.[1,2]

When sacred cows are challenged, much criticism can be expected. As was the case with the NBSS. Miller and colleagues defended the NBSS calmly and logically[3,4] and suggested that follow-up should continue until 1995 at least to confirm that the initial findings persisted through 10 years of follow-up.

How can one reconcile earlier diagnosis by mammography with no corresponding decrease in the death rate? Given the mind-set that earlier diagnosis must equate with an improvement in mortality (which sounds logical), I can appreciate the dismay that is engendered by the NBSS results.

I am a firm advocate of mammography, but like many good tests it has its limitations. Diagnostic mammography is especially useful in women over 40 years of age and when used in association with a competent CBE. The clinical assessment of a borderline change in the breast can be a most vexing diagnostic problem, even for the experience examiner. The dilemma with mammography arises when it is used as a screening tool, especially when it is used alone. Mammography will certainly pick up malignant lesions 2 to 3 years earlier than CBE alone. About two-thirds of these mammographic (occult) carcinomas are noninvasive or invasive but slow growing and have not metastasized. With the tests generally available, identifying these cancers a couple of years later by CBE apparently does not alter the death rate, based on the conclusion of the NBSS. Relating spread of breast cancer to distant body parts as a factor of time is an erroneous concept. Spread, however, is directly related to the host–tumour relationship, something over which we have no control at present.

The natural history of breast cancer suggests that many noninvasive cancers detected by mammography will not become invasive during the patient's lifetime. About one-third of cancers detected by mammography alone are invasive, with axillary-node involvement indicating systemic disease, even though distant metastases may not be demonstrated by current methods.

We would like to believe that if these cancers can be identified when the tumours are smaller than they would be if they were diagnosed later by CBE, the prognosis would be better because of the earlier introduction of chemotherapy or hormonal therapy. However, the NBSS found that although survival of patients whose disease was detected by mammography appears to be improved, the death rate is not.

The quality of mammography in the NBSS has been criticized. The landmark New York Health Insurance Plan Project (HIPP) published in 1988[5] used a state-of-the-art mammography that was certainly inferior to that used in the NBSS. The HIPP study showed a 30% to 40% reduction in mortality, especially in women aged 50 years and older, and it was the first scientific study to show the benefit of breast screening with CBE and mammography. The HIPP study was not designed to demonstrate the advantage of mammography over CBE in terms of reduction in the death rate. It was assumed that the chief contribution of screening in reducing the death rate was due to mammography. Could the competent CBE done in the HIPP study have been a major factor in the improved survival?

On the basis of the conclusions reached by the NBSS, Miller and colleagues advocated competent CBE as a sole method of breast screening in underdeveloped countries.[6,7] Miller has attempted to initiate such a program through the World Health Organization, but although there has been interest, sufficient funding has not yet been forthcoming (Miller AB: Personal communication, 1993).

We should make every effort at the medical school level to improve the skills of students in the perfor-

mance of a CBE. My experience in the first unit of the NBSS was that the CBE was generally done in a suboptimal manner by practising physicians. The comment most often heard from women who volunteered for the study was that the CBE done by the specially trained nurse-examiners was clearly superior to that provided by their physicians.

I confess that I was uncomfortable and disappointed by the conclusions reached in the NBSS. But then I was not comfortable when I began to perform modified radical mastectomy because I had been trained to regard radical mastectomy as the only appropriate surgical treatment. To think or do otherwise was heresy, and I well remember a prominent colleague whose university status was put in jeopardy because he advocated modified radical mastectomy as a proper substitute for radical mastectomy. My comfort level was again shaken when I began to do partial mastectomy. Controlled scientific studies in Canada, the United States, the United Kingdom and Europe attest to the equal results of partial as compared with radical mastectomy.

In conclusion, breast screening has been shown to reduce the death rate significantly, especially in women aged 50 to 69 years. The answer to the question of how significant is mammography as a screening tool awaits another couple of years' follow-up. In the meantime we must make every effort to improve the teaching of CBE in medical schools and hold ongoing teaching clinics for practising physicians.

We need a method by which we can discover breast cancer much earlier than we can with present-day mammography. A 2- to 3-year lead time in diagnosis is not sufficient to reduce the death rate. The final answer may well be a diagnostic test that will act like a smoke detector, telling us when and where cells are becoming dysfunctional. The sequel to that will be a method of favourably altering the tumour–host relationship, or the answer could equally well come from improved therapy for those with incipient distant metastases.

The postscript to the NBSS is eagerly awaited, and if the earlier conclusions stand up, we shall have to reconsider our approach to breast screening in light of economic restraints. Until then we should maintain but not enlarge breast screening programs.

Alan A. Bassett, MD, FRCSC, FACS
626–One Benvenuto Place
Toronto, ON
M4V 2L1

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Interpretation of Breast Biopsies

To the editors. As a pathologist with a special interest in breast lesions, I have noticed an increasing problem in the microscopic evaluation of breast biopsy specimens, particularly small ones. The problem is the result of what I presume to be a change in surgical technique. There is often significant coagulation necrosis at the margins of the specimen, which I interpret as originating from the use of cautery for excision. The coagulation necrosis distorts the cellular appearances to the extent of rendering them uninterpretable. Occasionally, I have been forced to report "No diagnosis possible" because of this problem.

Increasing reliance on mammographic findings has led to a greater proportion of small biopsies and a range of microscopic appearances with which pathologists are gradually becoming familiar. When performing a breast biopsy, the surgeon should bear in mind the importance of preserving the tissue and cell structure on which the diagnosis will be based.

Harry E. Emson, MA, MD, FRCPC
Professor of pathology
Department of Pathology
Royal University Hospital
Saskatoon, SK
S7N 0X0

The Angelchik Antireflux Prosthesis: Long-Term Clinical and Technical Follow-up

Claude Thibault, MD;* Picard Marceau, MD, MSc, PhD, FRCSC;† Simon Biron, MD, FRCSC;‡
Roch-André Bourque, MD, FRCSC;† Louis Béland, MD, FRCSC;§ Martin Potvin, MD, FRCSC‡

Objective: To evaluate the long-term clinical outcome and compare the rupture rate of the two generations of the silicone Angelchik antireflux prosthesis.
Design: A cohort study. Follow-up ranged from 61 to 119 months.
Setting: A university teaching hospital.
Patients: Sixty-three patients: 33 patients received the first generation Angelchik device (group 1) and 30 patients received a second generation design (group 2). The two groups were comparable for sex ratio, mean age and duration of symptoms.
Interventions: Implantation of the Angelchik prosthesis.
Main Outcome Measures: Comparison of the rupture rate and migration of the prosthesis as assessed by patient questionnaire, telephone interview and radiography of the abdomen.
Results: The prosthesis remained in a good position in 53% of group 1 patients and 93% of group 2 patients. The prosthesis was removed in 40% of group 1 patients, most often for rupture, and in only 7% of patients in group 2, to relieve dysphagia ($p < 0.01$). Grading on a Visick scale demonstrated a good result in 37% of group 1 patients and 69% of group 2 patients. Long-term dysphagia was the most prevalent adverse effect, seen in 45% of patients whose prosthesis was in a good position, and symptomatic reflux recurred in 8%.
Conclusions: The second generation of the Angelchik prosthesis, resulted in a reduced rupture rate of the prosthesis. Nevertheless the high complication and the failure rates militate against continued implantation of the prosthesis.

Objectif : Évaluer les résultats cliniques à long terme et comparer les taux de ruptures de deux générations de prothèses antireflux en silicone Angelchik.
Conception : Une étude de cohortes. La période de contrôle s'étend de 61 à 119 mois.
Contexte : Un hôpital d'enseignement universitaire.
Patients : Soixante-trois patients au total : 33 qui ont reçu l'instrument Angelchik de première génération (groupe 1) et 30 qui ont reçu le concept de seconde génération (groupe 2). Les deux groupes étaient comparables pour la répartition des sexes, la moyenne d'âge et la durée des symptômes.
Intervention : Pose de la prothèse Angelchik.
Principaux effets mesurés : Comparaison des taux de rupture et de migration des prothèses établis par questionnaire a'administré au patient, entrevue téléphonique et radiographie de l'abdomen.

From the Department of Surgery, Hôpital Laval, Sainte-Foy, Que.

**Resident in general surgery, †Professor of surgery and ‡Clinical professor, Department of Surgery, Université Laval, Hôpital Laval, Quebec*

§Clinical professor, Department of Surgery, Université Laval, Centre Hospitalier de l'Université Laval, Quebec

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Reprint requests to: Dr. Martin Potvin, Département de chirurgie, Hôpital Laval, 2725 Chemin Sainte-Foy, Sainte-Foy, QC G1V 4G5

Résultats : La prothèse est demeurée en bonne position chez 53 % des patients du groupe 1 et chez 93 % des patients du groupe 2. La prothèse fut retirée chez 40 % des patients du groupe 1, le plus souvent à cause d'une rupture, et chez seulement 7 % des patients du groupe 2, pour corriger une dysphagie ($p < 0,01$). L'évaluation à l'échelle de Visick a révélé de bons résultats chez 37 % des patients du groupe 1 et chez 69 % des patients du groupe 2. La réaction indésirable la plus fréquente a été une dysphagie de longue durée observée chez 45 % des patients dont la prothèse était bien positionnnée, et un reflux symptomatique dans 8 % des cas.
Conclusions : La deuxième génération de la prothèse Angelchik a entraîné une réduction du taux de rupture de la prothèse. Néanmoins, le haut taux de complications et d'échecs milite en faveur de l'abandon de cette prothèse.

The Angelchik prosthesis (Mentor Corp., Goleta, Calif.) was introduced as a surgical treatment for gastroesophageal reflux in 1979.[1] This horseshoe-shaped device made of silicone is tied around the distal esophagus beneath the diaphragm during a short, uncomplicated procedure.

Spectacular complications were reported in early trials, mostly related to rupture of the tie strap and migration of the prosthesis.[2–5] In January 1982, the strap design of the device was modified to solve that problem. The incidence of complications seems to have decreased since then,[6] but the manufacturer is the source of the supporting data.

In this cohort study of two generations of the Angelchik antireflux prosthesis we compared the rupture rate and migration of the device, and we report on the long-term clinical follow-up in 63 patients who received the Angelchik antireflux prosthesis.

Patients and Methods

The Prosthesis

The Angelchik prosthesis is a horseshoe-shaped implant consisting of a highly cross-linked silicone gel encased in a silicone elastomer shell to which a Dacron-reinforced silicone tie strap is attached. A tantalum radiopaque marker on the strap allows for easy roentgenographic localization. The tie straps in the initial design were attached to each extremity of the prosthesis and were prone to rupture by tearing downward. A circumferential strap was introduced in January 1982.

Procedure

The surgical approach was through an upper midline incision. After exploration of the peritoneal cavity, the diaphragmatic hiatus was exposed and the esophagus mobilized with minimal dissection. The device was passed around the esophagus, and the straps were tied anteriorly and secured with a large metallic clip. When the esophageal hiatus admitted more than two fingers, the diaphragmatic crura were approximated with silk sutures. Five surgeons at the same institution performed the operation in a similar manner.

Study Design

Between April 1980 and April 1986, 63 patients underwent implantation of the Angelchik prosthesis at Hôpital Laval, Sainte-Foy, Que.: 33 patients received the initial design of the device (group 1) and, after January 1982, 30 patients received the modified design (group 2). The indication for surgery was reflux esophagitis, based mostly on clinical symptoms, failure of medical treatment, endoscopic findings and results of reflux studies available at that time. In April 1986, because a mounting number of complications were being reported in the literature, we discontinued the use of the device.

Medical records of the patients were reviewed, and preoperative symptoms, surgical complications and recurrent symptoms were noted. A questionnaire was sent to every patient. Fifty-one (81%) questionnaires were completed and returned by mail and 5 others (8%) were filled by telephone interview. The remaining seven patients (11%) were identified as deceased. Review of the questionnaires and telephone interviews was carried out by the same observer. Radiographic films of the abdomen (anteroposterior and lateral) were obtained in all patients whose prosthesis had not been removed.

The clinical results were graded using a modified Visick scale as follows: 1—no symptoms, 2—mild symptoms not requiring treatment, 3—symptoms requiring, but controlled by, medical treatment and 4—symptoms uncontrolled by medical treatment or complications of the procedure.

Statistical Analysis

The p values were calculated by the χ^2 test, and Yates' correction for continuity[7] was applied to all calculations, according to the recommendations of Cochran[8] for small groups.

Follow-up

The follow-up ranged from 61 to

199 months (mean for group 1, 106 months and for group 2, 80 months).

Findings

Preoperatively, patients in the two groups were comparable with respect to sex, mean age and mean duration of symptoms. The mean follow-up was the only difference between the groups (Table I).

There was one death related to the procedure (2%). A 68-year-old woman was admitted to hospital because of fever and upper abdominal pain 3 months after an uncomplicated prosthesis implantation. Esophagoscopy showed that the prosthesis had eroded into the esophagus. The device was removed immediately, and the defect was closed primarily. Postoperatively, sepsis persisted, and the patient suffered multiple organ failure. She died 4 months after the second operation. Four patients in group 1 and two in group 2 died of causes unrelated to the prosthesis more than 3 years after their operation (Table II). Of these, one patient from group 1 survived an operation to remove a ruptured prosthesis.

Table I. Demographic Features of 63 Patients Who Had Placement of the Angelchik Antireflux Prosthesis

Demographic feature	Group 1, *n* = 33	Group 2, *n* = 30
Sex ratio, M/F	14/19	13/17
Mean age (and range) at surgery, yr	56.7 (25 – 83)	57.8 (20 – 81)
Mean duration of symptoms before surgery, mo	84	73
Medical treatment for more than 6 mo, no.	23	21
Previous antireflux surgery, no.	2	3
Investigation, no.		
pH metry	0	1
Barium transit	23	26
Manometry	24	21
Isotopic reflux study	10	15
Esophagoscopy	29	27
Biopsy	18	19
Mean follow-up (range), mo	106 (99 – 119)	80 (61 – 97)

*Group 1 received initial prosthesis design, group 2 received modified design.

Table II. Causes of Death

Patient no.	Group	Sex	Age, yr	Time after surgery, yr	Cause of death
1	1	F	68	0.6	Esophageal fistula, sepsis, ARDS
2	1	F	67	4	Metastatic adenocarcinoma of pancreas
3	1	M	30	5	Trauma
4	1	M	73	3	Metastatic carcinoma of lung
5	1	M	89	6	Metastatic neoplasia of unknown origin
6	2	F	74	3	Cardiac failure
7	2	M	78	7	Sudden death at home

ARDS = acute respiratory distress syndrome

Table III. Follow-up Findings in Patients Receiving the Angelchik Prosthesis With Respect to Migration and Rupture of the Prosthesis

Finding	Group 1, no. (%), *n* = 30*	Group 2, no. (%), *n* = 28*	*p* value
Good position	16 (53)†‡	26 (93)‡	< 0.01
Migration	2 (7)	1 (4)	NS
Rotation	2 (7)	0	NS
Rupture	10 (33)	1 (4)	< 0.01
Erosion in gastrointestinal tract	2 (7)	0	NS

Mean follow-up in group 1 was 106 months and in group 2 was 80 months.
*Patients who died were excluded unless the prosthesis had been removed before death.
†Two patients had a previously ruptured prosthesis replaced with one of the same type.
‡Two patients had their prosthesis removed because of dysphagia.

The initial design prosthesis (group 1) was shown to be in good position in 16 (53%) of 30 patients at a mean follow-up of 106 months (Table III). In two of these patients a second prosthesis of the same design was inserted after the first one had ruptured. The second design of the prosthesis (group 2) was shown to be in good position in 26 (93%) of 28 patients at a mean follow-up of 80 months. In both groups, the prosthesis had been removed in two patients because of dysphagia but the devices were found to be in good position at operation.

The prosthesis migrated into an intrathoracic position in 2 (7%) of 30 patients in group 1 and 1 (4%) of 28 patients in group 2, but it remained around the esophagus. Although one of the group 1 patients was completely asymptomatic, the other two experienced recurrent reflux, which was controlled with medical treatment. Rotation of the device was observed in two (7%) patients of group 1 and none of group 2.

Strap rupture of the prosthesis occurred in 10 (33%) group 1 patients and 1 (4%) group 2 patients ($p < 0.01$). In group 1, rupture was confirmed less than 36 months after the initial procedure in six patients and 78 months after implantation in one patient. All seven patients un-

derwent reoperation because of recurrent reflux. In two patients, the device was replaced with one of the same type; the other patients had a Nissen fundoplication. Another ruptured device was discovered incidentally 74 months after the initial operation, during an elective cholecystectomy. This prosthesis, like most of those examined surgically, was encapsulated in a fibrous sheath. The prosthesis was removed for fear of migration or erosion; although no antireflux procedure was performed, the patient has remained symptom free. Two intraperitoneally free prostheses identified radiographically in asymptomatic patients were likely the result of strap rupture. In one asymptomatic patient, the prosthesis could not be found after thoracic and abdominal roentgenographic evaluations. The prosthesis may have eroded into the esophagus or stomach.

In group 2 patients, only one case of rupture was suspected, in an asymptomatic patient. The abdominal films showed a correct esophagogastric position of the device, but the tips were abnormally separated.

The prosthesis was removed in 13 (39%) group 1 patients and 2 (7%) group 2 patients (Table IV). The mean time at reoperation was 34 months in both groups (range from 3 to 78 months in group 1 and 20 to 48 months in group 2). Reasons for removal included rupture, migration, rotation or erosion, recurrence of reflux and dysphagia (Table IV).

Clinical failure (Visick grade 4) was demonstrated in 18 (60%) patients of group 1 and 5 (18%) of group 2 (Table V). According to one (3%) patient of group 1 and four (14%) patients of group 2, there was no amelioration of symptoms, and medication was required to control their reflux symptoms (grade 3). Eleven (37%) of 30 group 1 patients and 19 (68%) of 28 group 2 patients had good results (grade 1 or 2) at a follow-up of 106 and 80 months respectively.

Dysphagia was the predominant postoperative symptom. It was noted in 17 (45%) of 38 patients whose prosthesis was in good position at the time of examination. Most of these patients experienced mild dysphagia, as evidenced by the need to ingest small pieces of food, masticate thoroughly and take fluid between swallows. This symptom was more pronounced in the 1st year after operation in only two (5%) patients, whereas the others experienced no modification of that symptom after the 1st year. One patient complained of an inability to swallow steak or raw vegetables (grade 4).

The second most frequent symptom was persistent or recurrent reflux. This was present in 13 (34%) of 38 patients with the prosthesis in a good position. Seven (18%) patients complained of mild occasional reflux (grade 2), and three (8%) patients took H_2 receptor blockers with good results (grade 3). In three (8%) patients the symptom was not relieved by medications (grade 4).

Pyrosis was reported in 11 (29%) of the 38 patients. In all cases it was mild or was relieved by medical treatment. Gas bloat syndrome, noted in seven (18%) patients, was mild. Twenty (53%) patients reported complete relief of their preoperative symptoms.

When we questioned the 38 patients whose prosthesis was in a good position about the degree of satisfaction with the procedure, 20 (53%) were very satisfied, 13 (34%) were satisfied, 4 (10%) were poorly satisfied and 1 (3%) was dissatisfied.

Table IV. Causes of Reoperation

Cause	Group 1, no. (%), *n* = 33	Group 2, no. (%) *n* = 30	*p* value
Rupture, migration, rotation or erosion	9 (27)	0	< 0.01
Reflux recurrence	2 (6)	0	NS
Dysphagia	2 (6)	2 (7)	NS
Total	13 (39)	2 (7)	< 0.01

Table V. Findings According to Visick Grade

Grade	Group 1, no. (%), *n* = 30*	Group 2, no. (%), *n* = 28*	*p* value
1	9 (30)	8 (28)	NS
2	2 (7)	11 (39)	< 0.01
3	1 (3)	4 (14)	NS
4	18 (60)	5 (18)	< 0.01

*Patients who died were excluded unless the prothesis had been removed before death.

Discussion

This comparison of two generations of the Angelchik antireflux prosthesis demonstrated that the first design had higher removal and reoperation rates and a worse outcome, as shown by a modified Visick score. It demonstrated that even if the position of the prosthesis was adequate, 47% of patients experienced one or more symptoms associated with the device.

This study was conducted to evaluate the clinical outcome after insertion of the prosthesis. It was not an objective assessment of reflux control. Other studies[9–12] have al-

ready proved the efficacy of the prosthesis in the control of gastroesophageal reflux. The main controversies have concerned the long-term removal rate and adverse symptoms, principally dysphagia.

The rate of prosthetic rupture was 33% (10 patients) in group 1, compared with only 4% (1 patient) in group 2. The removal rate was 40% for group 1 compared with 7% for group 2 and was related mainly to the difference in the rupture rate. The strap design of the prosthesis, modified in 1982, seems to have resulted in a decrease in the frequency of rupture. Although the follow-up in group 2 patients was shorter, the mean time to reoperation was identical in the two groups. The worst period for rupture was the first 36 months after implantation, but long-term risk persists as evidenced by a ruptured prosthesis 78 months after implantation. It was not possible to determine the exact time of rupture, and the reoperation time does not necessarily correlate with the rupture time.

In previous reports,[13–16] the removal rate of the prosthesis varied between 5% and 17%, but the mean follow-up rarely exceeded 36 months. Most studies have not differentiated between the two designs of prosthesis, even if the study period included the beginning of 1982. However, one paper[17] reported that 4 (14%) of 28 patients with the initial prosthetic design and no patients with the modified design experienced rupture necessitating removal of the prosthesis during a 5-year period.

With respect to patients with the prosthesis in a good position, dysphagia was the predominant symptom in 17 (45%). This complication has been reported to be transitory and to last less than 6 months after operation.[17,18] In our series, most patients experienced no improvement of their dysphagia over time, but they have adapted to this problem by changing their eating habits. The same conclusion was reached in other studies, in which a relative long-term dysphagia rate of 10% to 96% was reported.[10,16,19] In a recent prospective, randomized trial of Nissen fundoplication versus implantation of the Angelchik prosthesis, 5 of 25 patients who received the prosthesis experienced short-term dysphagia and 3 required removal of the device.[20] Another study reported that in four patients (23%), all of whom were classified as having Visick grade 1 symptoms at 2 years, intermittent dysphagia developed between 4 and 5 years postoperatively.[21] The rotation/displacement complication has been described to explain some, but not all, cases of dysphagia.[15,17] We hypothesized that the prosthesis was not distensible enough or that the fibrous reaction that occurs around the prosthesis may contribute to dysphagia.

Although only 20 (53%) of the patients with their prosthesis in a good position reported complete relief of preoperative symptoms, 33 (87%) were subjectively satisfied or very satisfied with their operation. This suggests that postoperative symptoms were less disturbing than the preoperative symptoms.

Only 19 (68%) group 2 patients had a good result on modified Visick grading. In a randomized trial,[22] a good Visick grade was obtained at a mean follow-up of 38 months in 77% of the patients receiving the Angelchik prosthesis compared with 94% of the patients who underwent a Nissen fundoplication. In contrast, another randomized study[23] demonstrated a good Visick grade in 96% of patients receiving the Angelchik prosthesis compared with 81% of the patients who underwent a Nissen fundoplication during a 2-year period. However, Timoney, Kelly and Welfare[24] reported a change in Visick grading over time, from 92% with a good result at a follow-up of 2 to 23 months, to 71% at a follow-up of 14 to 35 months.

Conclusions

The modified Angelchik prosthesis has an acceptable rupture rate at a mean follow-up of 80 months, but the adverse symptoms seem unacceptably high. Therefore, we do not advocate the use of the prosthesis in its present form for the prevention of reflux esophagitis.

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SESAP VII Question / Question SESAP VII

Items 82 to 84

(A) Computed tomographic (CT) scan
(B) Radioisotope biliary excretion scan
(C) Magnetic resonance imaging (MRI)
(D) Endoscopic retrograde cholangiopancreatography (ERCP)
(E) Ultrasonography

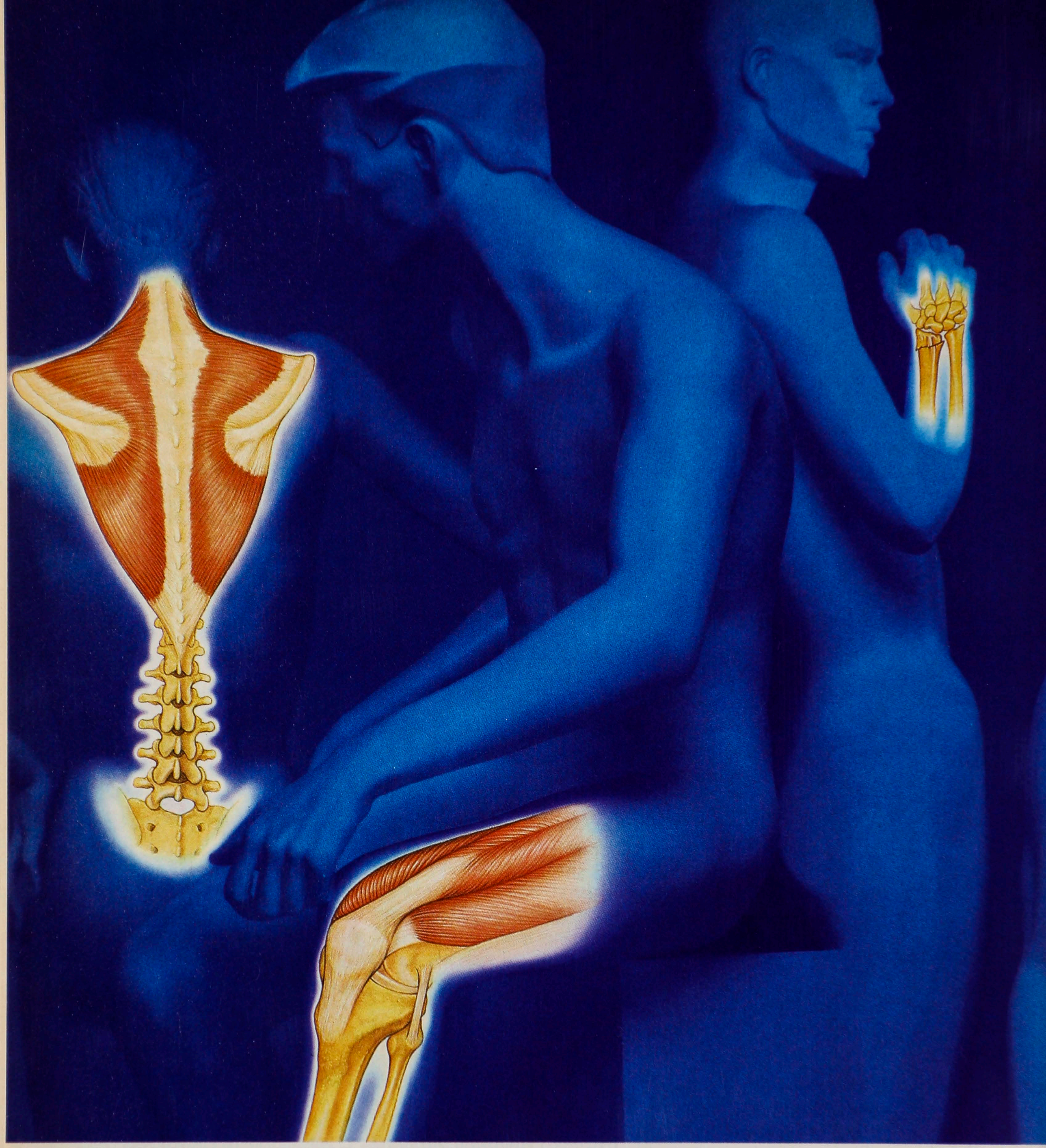
82. Most effective study for diagnosis of retained stones in the biliary ductal system

83. Preferred initial study for a patient who has jaundice with an apparent obstructive origin

84. Preferred follow-up study in a patient with clinical signs and symptoms of acute cholecystitis and no stones in the gallbladder by ultrasonography

For the numbered completions above select the one lettered heading that most closely associated with that number. Each letter may be selected once, more than once or not at all.

For the critique of Items 82 to 84 see page 42.



Emergency Medicine[22,23,57]

Renal colic

Low back pain

Sprains and strains

Orthopedic Surgery[4-9,48,55]

Total hip or knee replacement

Open reduction and fixation of long bone fractures

Meniscectomy

Trauma[9,22]

Fracture pain

Acute trauma pain

Contusions and lacerations

TORADOL IM

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Toradol IM offers an improved side effect profile compared to narcotics.[5,16] Patients generally experience a lower incidence of nausea, vomiting, constipation, drowsiness, and respiratory depression.[5,22,55,57] As a result, Toradol patients usually benefit from a faster return to normal activities and earlier discharge.[13]

Toradol tablets provide effective and generally well-tolerated analgesia when used alone or as follow-on therapy to Toradol IM.

By class, Toradol belongs to the non-steroidal anti-inflammatory drug (NSAID) family. However, unlike conventional NSAIDs, it is a potent analgesic with minimal anti-inflammatory and antipyretic activity.

As with all NSAIDs, the most common side effects with Toradol involve the G.I. tract. Toradol is contraindicated in patients with peptic ulcer, active inflammatory disease of the G.I. system and patients who display hypersensitivity to the drug itself, ASA, or other NSAIDs.[2]

A highly effective[4-10] therapy, Toradol IM gives patients the advantages of non-narcotic analgesia in the short-term treatment of moderate to severe acute pain.

General Surgery[4-9,59-61]

Cholecystectomy
Gastric bypass
Thoracotomy
Hernia repair

Gynecological Surgery[9,38,39,46,59]

Abdominal hysterectomy
Vaginal hysterectomy
Laparotomy
Post-partum uterine cramping

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10 mg tablets & 30 mg/mL IM injections KETOROLAC TROMETHAMINE

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For prescribing information see page 62

Cystic Hydatid Lung Disease in Northwest Canada

Randy D. Moore, MD; John D. Urschel, MD, FRCSC, FRCSEd; Ronald E. Fraser, MB, ChB, FRCSEd; Someshwar S. Nakai, MB, BS, FACS, FRCSC; Albert J. Geeraert, MD, FACS, FRCSC

Objective: To determine the optimal treatment of sylvatic cystic hydatid lung disease.
Design: Retrospective case study.
Setting: Five Edmonton hospitals serving northern Alberta and parts of the Northwest Territories.
Patients: Fourteen patients with cystic (*Echinococcus granulosus*) hydatid lung disease.
Interventions: Cyst enucleation, wedge resection and pulmonary lobectomy.
Main Outcome Measures: Eradication of pulmonary hydatid disease and complications of treatment.
Results: Mean patient age was 32 years. Eight patients were symptomatic. Liver cysts were present in three patients. One of the three patients managed by observation required surgery for an expanding cyst. Surgery (13 procedures in 12 patients) was successful in eradicating pulmonary hydatid disease: 8 cyst enucleations, 3 wedge resections and 2 lobectomies were done. There was only one major postoperative complication (pneumatocele requiring repeat surgery). Intraoperative cyst rupture occurred in five cases, but anaphylaxis or seeding did not result. There were no bronchopleural fistulae.
Conclusions: Sylvatic cystic hydatid lung disease is more benign than pastoral hydatid disease. Patients with asymptomatic cysts should be managed by observation. Surgery, consisting of endocyst enucleation or wedge resection, is indicated for symptomatic, enlarging or infected cysts.

Objectif : Établir quel est le traitement optimal de l'hydatidose pulmonaire d'origine sylvestre.
Conception : Une étude rétrospective de cas.
Contexte : Cinq hôpitaux d'Edmonton desservant le Nord de l'Alberta et certaines parties des Territoires du Nord-Ouest.
Patients : Quatorze patients souffrant d'hydatidose pulmonaire (*Echinococcus granulosus*).
Interventions : Énucléation kystique, résection cunéiforme et lobectomie.
Principaux effets mesurés : L'éradication de l'hydatidose pulmonaire et les complications de traitement.
Résultats : L'âge moyen des patients était de 32 ans. Huit patients étaient symptomatiques. Des kystes hépatiques étaient présents chez trois patients. Un des trois patients mis sous simple observation nécessita une intervention chirurgicale due à l'expansion du kyste. La chirurgie (13 interventions chez 12 patients) réussit à éliminer l'hydatose pulmonaire : on a pratiqué 8 énucléations kystiques, 3 résections cunéiformes et 2 lobectomies. On n'a enregistré qu'une seule complication postopératoire majeure (un pneumatocèle nécessitant une deuxième opération). Une rupture peropératoire d'un kyste survint à cinq occasions mais celle-ci n'entraîna ni anaphylaxie, ni propagation. Il n'y eut aucune fistule bronchopleurale.
Conclusions : L'hydatidose pulmonaire d'origine sylvestre est plus bénigne que celle d'origine champêtre. Les patients porteurs de kystes asymptomatiques doivent être traités par simple observation. La chirurgie consistant en une énucléation ou une résection cunéiforme des kystes est indiquée quand il s'agit de kystes symptomatiques, évolutifs ou infectés.

From the Department of Surgery, The University of Alberta, Edmonton, Alta.

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Reprint requests to: Dr. John D. Urschel, Department of Surgical Oncology, Roswell Park Cancer Institute, Elm and Carlton Sreets, Buffalo, NY 14263-0001, USA

Cystic hydatid lung disease occurring in northwest Canada appears to be different from hydatid lung disease seen elsewhere in the world in that it involves lung more often than liver, has a relatively benign natural history and is associated with wild natural hosts. The term "sylvatic" has been applied to the northwest Canadian form of hydatid disease. To determine the optimal treatment of sylvatic cystic hydatid lung disease we reviewed our recent experience with this condition.

Methods

We reviewed the records of patients treated for cystic hydatid lung disease in five Edmonton hospitals between 1970 and 1992. The *International Classification of Diseases*, 9th revision (ICD-9-CM) was used. The diagnosis was established by pathologic means in patients who underwent resection and by a combination of epidemiologic history, positive serologic findings and suggestive findings on chest radiography in patients treated nonoperatively. Patients with alveolar hydatid disease due to *Echinococcus multilocularis* infection were excluded. Details of the clinical presentation, diagnostic methods and treatment and the final outcome were recorded.

Findings

Thirty-five patients were admitted to Edmonton hospitals with cystic hydatid lung disease between January 1970 and July 1992. Twenty-one patients whose records were incomplete were excluded, leaving 14 patients available for study.

The 14 patients (9 male, 5 female) ranged in age from 3 to 65 years (mean 32 years). Race was not routinely recorded, but many patients had surnames that suggested an aboriginal origin. Geographic and animal host factors were compatible with sylvatic cystic hydatid lung disease in 13 patients; 1 patient had previously lived in the Middle East and may have had the pastoral type of hydatid disease. The disease was confined to the lungs in 11 patients and involved both lung and liver in 3 patients.

Pulmonary cysts caused symptoms in eight patients. The symptoms included chest pain (six patients), cough (three patients), dyspnea (two patients) and fever (one patient), alone or in combination. Eosinophilia was present in six patients. Results of the Casoni test were positive in five of seven patients tested. Serologic tests (latex agglutination, indirect hemagglutination or enzyme-linked immunosorbent assay [ELISA]) gave positive results in three of six patients tested. Chest radiographs were abnormal in all patients. Eleven had solitary cysts and 3 had multiple cysts. Lobar distribution was even (right upper lobe 5, right middle lobe 1, right lower lobe 5, left upper lobe 4, left lower lobe 4).

Three patients were initially managed by observation; one of these patients eventually required operation. Twelve patients underwent 13 operations. Hydatid lung disease was diagnosed preoperatively in 6 of 12 patients. The indications for operation included an undiagnosed mass on chest radiography in six patients, a symptomatic cyst in three patients, an expanding cyst in one patient, an infected cyst in one patient and uncomplicated hydatid cyst in one patient. Surgical procedures consisted of cyst enucleation (eight patients), wedge resection (three patients) and lobectomy (two patients). The diameter of the resected cysts ranged from 3 to 15 cm (mean 6 cm).

There were no operative deaths. Intraoperative cyst rupture occurred during five resections, but there was no echinococcal seeding or anaphylaxis. Two patients had postoperative complications: in one patient with an infected cyst, a large pneumatocele developed after enucleation and capitonnage (a suturing technique that obliterates the residual pulmonary cavity), and lobectomy was required; in the second patient superficial venous phlebitis developed secondary to inappropriate antibiotic administration. There were no bronchopleural fistulas or prolonged air leaks. Mean duration of chest tube drainage was 4 days. Postoperative hospitalization ranged from 4 to 14 days (mean 10 days).

All 12 patients treated surgically had satisfactory results. No recurrent cysts have been encountered, but the follow-up has been difficult. Many patients live a great distance from Edmonton. Of the three patients managed initially by observation, the condition of two who had small cysts remained stable, but the follow-up period was less than 3 years. One patient's cyst expanded during the period of observation, and operation was required.

Discussion

Echinococcus granulosus is the organism responsible for cystic hydatid lung disease, and *Echinococcus multilocularis* causes alveolar hydatid lung disease.[1] Cystic hydatid disease is the most common form encountered in North America. It comprises two subsets that differ in natural history, organ involvement and natural host.[2] Pastoral hydatid disease involves liver more often than lung, is prone to serious complications and is associated with domestic natural hosts. Sylvatic hydatid disease involves lung more often than liver, has a relatively benign natural history and is associ-

ated with wild natural hosts.[2,3] The wolf is the natural definitive host, and large ungulates, such as moose, caribou and reindeer, are intermediate hosts. Dogs serve as alternative definitive hosts.

The benign nature of sylvatic cystic hydatid lung disease was confirmed in this study. Only one of our patients presented with secondary cyst infection. Spontaneous intrapleural or bronchial rupture with dissemination or anaphylaxis did not occur. Even intraoperative cyst rupture proved innocuous in this series. The low postoperative morbidity and mortality in our series relative to those of large published surgical series of pastoral hydatid lung disease[4,5] are probably a reflection of severity of underlying disease rather than operative skill.

The diagnosis of cystic hydatid lung disease is now usually established without resorting to diagnostic thoracotomy. In patients with a history of exposure to definitive animal hosts in northwest Canada, positive serologic findings and a cystic pulmonary lesion on chest radiography are diagnostic. The Casoni skin test is no longer used, because its sensitivity and specificity are low. The ELISA is the most sensitive of the commonly available serologic tests,[6] but it appears to be more useful in pastoral hydatid disease. Serologic tests in our series often gave negative results; others[7] have noted a low yield of positive results on serologic testing in sylvatic hydatid disease. Therefore, the combination of a compatible epidemiologic history and a cystic lesion on chest radiography is probably sufficient to make a tentative diagnosis of sylvatic cystic hydatid lung disease.

The standard treatment of pastoral cystic hydatid lung disease is surgical resection, but the indications for surgery in sylvatic hydatid disease are less clearly defined.[2,4,8] We recommend surgery for symptomatic, infected or enlarging pulmonary hydatid cysts. Thoracotomy may also be required in cases of diagnostic uncertainty. Asymptomatic sylvatic hydatid cysts can be observed. Many remain uncomplicated and asymptomatic.[2,3]

We have no experience with albendazole or mebendazole in the treatment of sylvatic pulmonary hydatid disease. The role of medical therapy, if any, has not been defined for this northwest Canadian form of hydatid disease. In pastoral cystic hydatid disease, albendazole is recommended as an adjunctive measure in selected surgical patients and as the sole treatment for patients who are not candidates for surgery. Albendazole is recommended for most patients with alveolar hydatid disease.[1,9]

Surgical treatment for sylvatic cystic hydatid disease should be as conservative as possible. Endocyst enucleation, with or without capitonnage of the residual cavity, was the commonest procedure in our series. Capitonnage is a suture technique that obliterates the residual pulmonary cavity.[8,10] Intraoperative cyst rupture was quite common during enucleation. Fortunately, cyst rupture is essentially harmless in the sylvatic form of the disease.[3] The fact that the sylvatic cyst wall is more delicate than the pastoral cyst wall accounts for the high incidence of intraoperative rupture in sylvatic hydatid surgery.[2] Therefore, wedge resection has been advocated[2] Our experience with wedge resection for small cysts was favourable, but wedge resection of large cysts is difficult, so enucleation is preferable. Lobectomy should be reserved for patients with multiple intralobar cysts, giant cysts or extensive lobar destruction resulting from infection.

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Chronic, Contained Rupture of Aortic Aneurysms Associated With Vertebral Erosion

Paul F. Galessiere, MD;* Allan R. Downs, MD, FRCSC;* Howard M. Greenberg, MD, FRCPC†

Chronic, contained rupture of aortic aneurysms is an important subset of ruptured aneurysms. Their presentation is unusual, and the diagnosis may be delayed. The associated vertebral erosion may contribute to the difficulty in diagnosis. The courses of three patients with chronic, contained rupture of an aortic aneurysm associated with vertebral erosion are presented. In each case the presentation was unusual and the initial diagnosis delayed. Computed tomography demonstrated the contained rupture and vertebral erosion in all cases. Microorganisms were cultured from all three aneurysms. Repair was successful in all cases. Contained rupture of an aortic aneurysm should be considered in older patients with unexplained back pain, and the possibility of this condition should not be ignored when there is a history of arthritic back pain or a radiograph suggestive of degenerative disease. Urgent surgical repair is indicated to prevent lethal, uncontrolled hemorrhage.

Les ruptures chroniques, cloisonnées d'anévrismes aortiques constituent un sous-groupe important des ruptures d'anévrismes. Leur tableau clinique est inhabituel et leur diagnostic peut être retardé. L'érosion vertébrale qui leur est reliée peut contribuer aux difficultés de diagnostic. On décrit l'évolution de trois patients ayant souffert de rupture chronique, cloisonnée d'un anévrisme aortique associée à une érosion vertébrale. Dans chaque cas, le tableau était inhabituel et le diagnostic initial fut retardé. Dans tous les cas, la tomographie par ordinateur permit de mettre en évidence la rupture cloisonnée et l'érosion vertébrale. Des microorganismes furent isolés des trois anévrismes. La réparation fut réussie dans tous les cas. Une rupture cloisonnée d'un anévrisme aortique doit être envisagée chez les patients âgés souffrant de maux de dos inexpliqués et la possibilité d'une telle affection doit être gardée à l'esprit quand il y a des antécédents de douleurs arthritiques dorsales ou quand les radiographies sont indicatives d'une maladie dégénérative. Une réparation chirurgicale urgente s'impose pour prévenir que ne survienne une hémorragie mortelle.

The entity of chronically contained aortic rupture is undoubtedly as old as the history of the aortic aneurysm. However, it has only been fully described in the literature since the advent of computed tomography (CT). Its presentation is distinctly different from that of acute rupture of an aortic aneurysm. Patients with chronically contained rupture are hemodynamically stable and have a long history of back pain with symptoms attributable to the compressive effects of a chronic retroperitoneal hematoma on retroperitoneal structures. Some symptoms are the result of vertebral erosion, which is a frequent associated finding. It is important to recognize this entity and to treat it promptly because, if it is left untreated, uncontrolled rupture is inevitable followed by death in most cases.

Three cases of chronic, contained rupture of an aortic aneurysm are the subject of this report. One pa-

*From the *Department of Surgery and †Department of Radiology, University of Manitoba, and the Health Sciences Centre, Winnipeg, Man.*

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Reprint requests to: Dr. Allan R. Downs, Department of Surgery, Health Sciences Centre, 820 Sherbrook St., Winnipeg, MB R3A 1R9

tient presented with distal embolization that had not previously been reported. All the patients had associated vertebral erosion, which added to the initial diagnostic difficulty. Definitive diagnosis was made by CT, and successful repair followed.

Case Reports

Case 1

A 58-year-old man, who had previously been healthy, experienced acute, severe lower back pain that radiated to his left thigh and calf 6 weeks before admission to hospital. The pain resolved within 48 hours, during which time he noted increasing numbness, discomfort and coolness of his left forefoot and toes. Initial consultation with his family doctor resulted in treatment for what was thought to be gout. Over the next few weeks there were progressive ischemic changes in his left forefoot. On re-evaluation, his family doctor noted a pulsatile abdominal mass and gangrenous changes to the patient's left toes.

On referral to our institution he did not complain of any back pain. He was normotensive, with a pulsatile abdominal mass and a partially gangrenous left forefoot. His hemoglobin level was 124 g/L, his leukocyte count was 9.3×10^9/L, and his erythrocyte sedimentation rate was 39 mm/h. The ankle brachial indices were normal.

We believed that this presentation represented distal embolization from an intact abdominal aortic aneurysm. Lateral x-ray films of the lumbar spine revealed what was initially interpreted as a compression fracture of the L2 vertebral body and a calcified aortic wall. CT revealed an 8-cm infrarenal abdominal aortic aneurysm, which had perforated posteriorly and caused extensive erosion of the L2 vertebral body. There was also a long-standing hematoma in the right parapsoas region, displacing the ureter and kidney to the right (Fig. 1A).

Forty-eight hours later, coincidental with the resurgence of his back pain, the patient was operated upon. On opening the aneurysm, we found a large defect in the posterior aortic wall, with erosion into the L2 vertebral body and intervertebral disc (Fig. 1B). The right parapsoas region contained old hematoma as well as some fresh clot. The aneurysm was repaired with an aortobifemoral Dacron graft.

Culture of the contents of the aneurysm and intervertebral space yielded a growth of *Escherichia coli*. The patient was treated with gentamicin (60 mg intravenously every 8 hours) and cefazolin sodium (1 g every 8 hours) for 4 weeks. A transmetatarsal amputation was performed 15 days after the aneurysm repair. The patient was discharged from hospital 1 month after the aneurysm repair. Cefazolin (250 mg every 6 hours) was prescribed for 3 months. A follow-up CT scan 2 years later showed a healed graft. The patient was well at follow up 5.5 years postoperatively.

Case 2

A 56-year-old normotensive man with chronic obstructive pulmonary

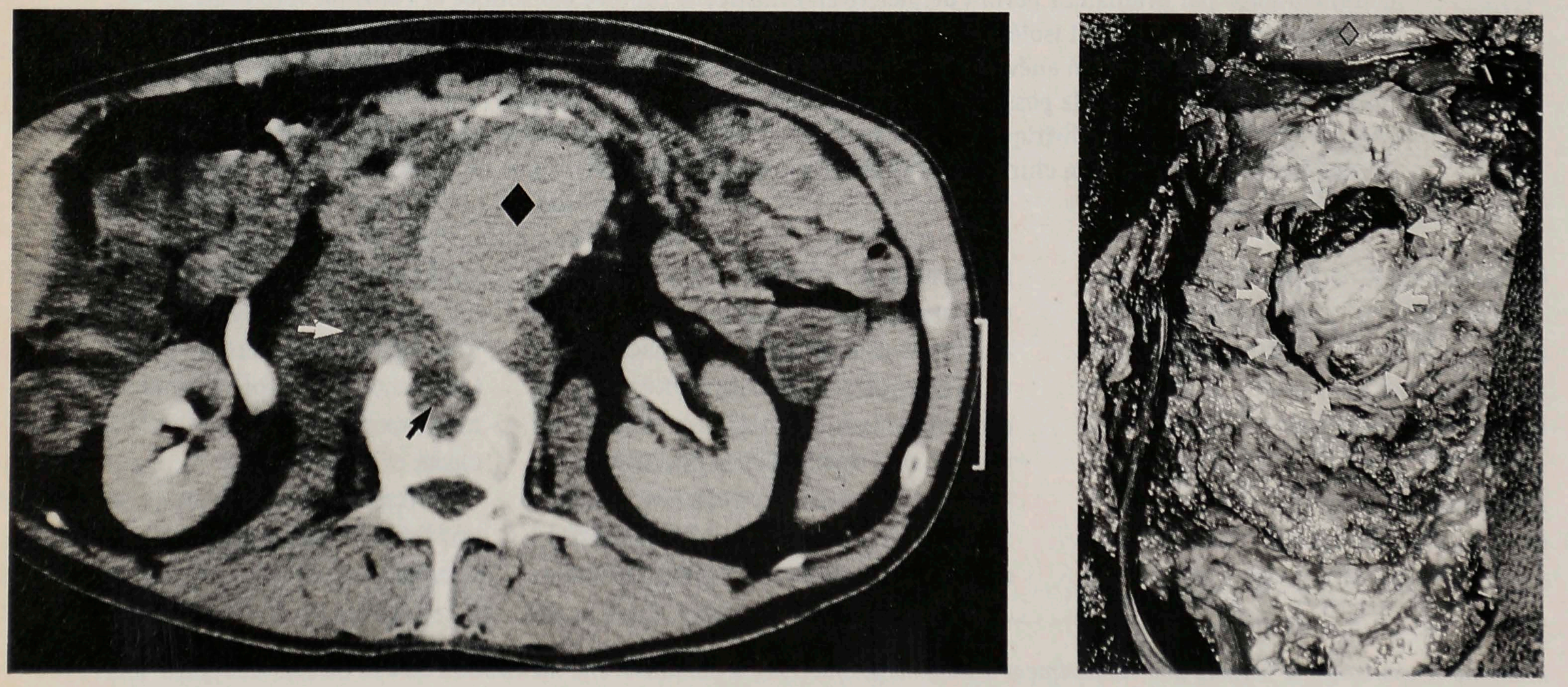

FIG. 1. Case 1. (Left) Computed tomography (CT) scan demonstrates ruptured abdominal aortic aneurysm (AAA) (diamond), right-sided retroperitoneal hematoma (white arrow) and vertebral body erosion (black arrow). (Right) Intraoperative view of opened aneurysm demonstrates defect in posterior wall (white arrows) with erosion into underlying vertebral body and disc. Left renal vein is seen at top (diamond).

disease and chronic back pain, which was attributed to long-standing osteoarthritis of his lumbar spine, had a 5-cm abdominal aortic aneurysm. While awaiting aneurysm repair he presented with peritonitis due to perforation of a sigmoid diverticulitis. He underwent a sigmoid resection with creation of a colostomy and mucous fistula. This was followed by pneumonia, acute respiratory distress syndrome, pelvic abscess and wound dehiscence. After a prolonged hospital stay he was discharged in good condition. At the time of discharge a CT of his abdomen revealed an intact infrarenal abdominal aortic aneurysm and degenerative changes in a lumbar vertebra (Fig. 2A).

The patient was seen again 6 weeks later, with a 2-week history of worsening lower abdominal and back pain. He was normotensive and had a body temperature of 37.6°C. He had a functioning colostomy and lower abdominal tenderness. His leukocyte count was 18.3×10^9/L with toxic granulation. His erythrocyte sedimentation rate was 100 mm/h, and his hemoglobin level was 135 g/L. Recurrent pelvic infection was considered, so he was started on imipenem (500 mg intravenously every 8 hours). CT (Fig. 2B) demonstrated that the aneurysm had enlarged slightly and had perforated posteriorly. The hematoma was contained by the vertebral body and extended into the retroperitoneal space around the psoas muscle. There was also evidence of erosion into the L3 vertebral body. No intra-abdominal sepsis was demonstrated. Gallium scanning failed to reveal any intra-abdominal source of infection.

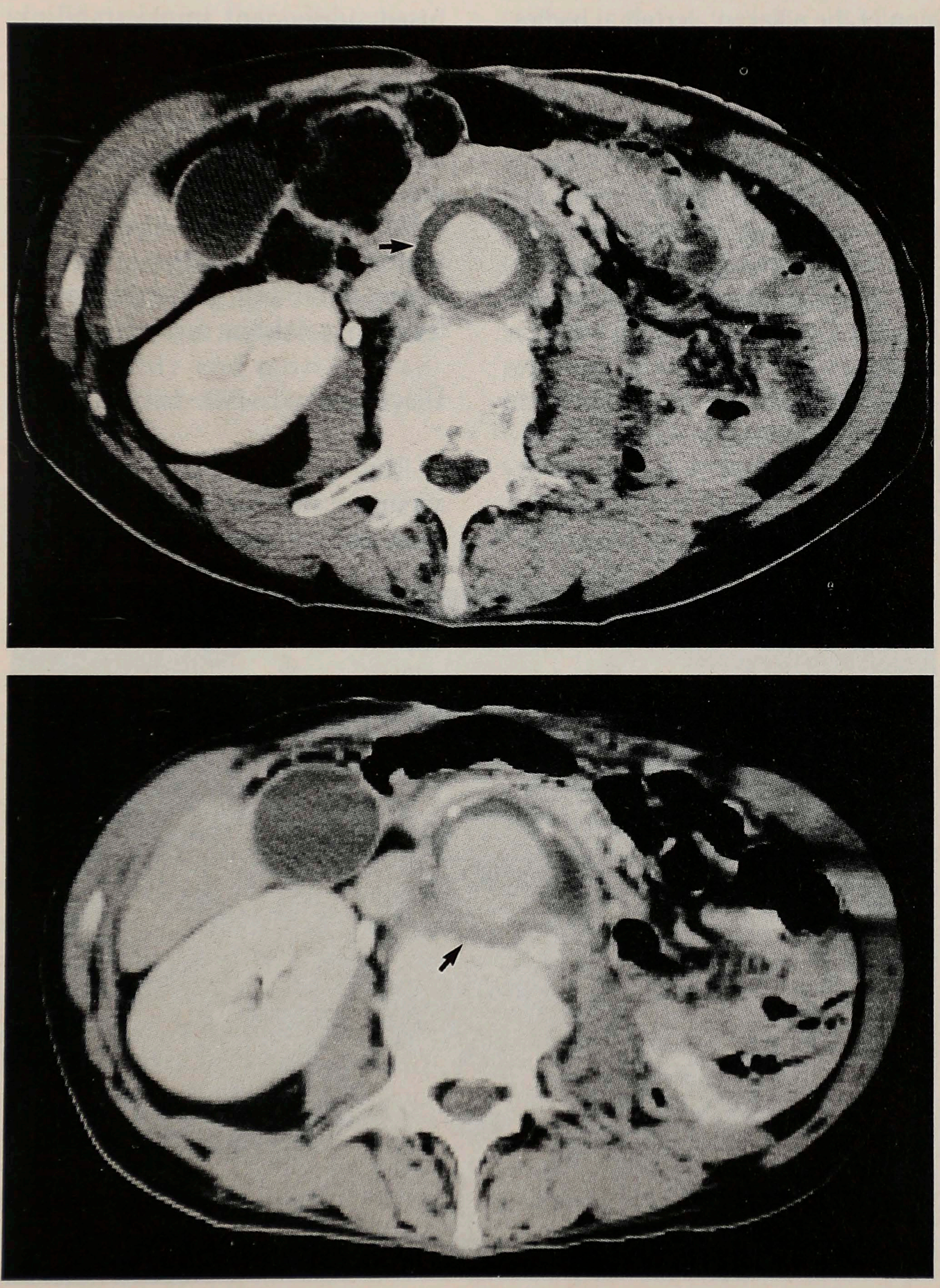

FIG. 2. Case 2. CT scan before (top) and after (bottom) rupture. Note intact AAA before rupture (arrow). In bottom illustration, contained rupture with erosion of vertebral body (arrow) can be seen.

At operation through a retroperitoneal approach with a thoracoabdominal incision, the aneurysm was found to have ruptured into the anterior portion of the L3 vertebral body and intervertebral disc. Temporary proximal control was obtained above the diaphragm. The aneurysm was replaced by a knitted Dacron tube graft. Gram's staining of the aortic contents was negative for organisms, but culture yielded a 1+ growth of *Staphylococcus epidermidis* and a 1+ growth of *Clostridium* sp. The patient had an uncomplicated postoperative course. He was treated with imipenem (500 mg intravenously every 8 hours) for 10 days and clindamycin (300 mg orally every 8 hours) for 12 weeks. On follow-up at 14 months, he had gained 18 kg. He had claudication of the right calf but otherwise was doing well. Aortography demonstrated a normal aortic graft and occlusion of the right superficial femoral artery.

Case 3

This 81-year-old man complained

of nonspecific mid-back pain for 2 years. The pain had worsened in the weeks before his admission to hospital. His appetite had decreased, and he had lost approximately 16 kg over several months. Ultrasonography revealed an aneurysm of the lower thoracic aorta, 5 cm in diameter, and underlying vertebral erosion just above the level of the diaphragm.

The patient smoked cigarettes but had no history of hypertension. When seen at our institution he was cachexic and afebrile. His blood pressure was 160/85 mm Hg, and he had normal pulse and respiratory rates. His abdomen was not tender, and an aneurysm was not palpated. He had an obvious thoracic kyphus and tenderness upon palpation of his lower thoracic spine.

Laboratory investigations revealed a hemoglobin level of 118 g/L, a leukocyte count of 8.0×10^9/L and an erythrocyte sedimentation rate of 34 mm/h. Thoracic spine x-ray films revealed vertebral body destruction of the T11 and T12 vertebrae, producing a slight gibbus deformity at the level of a calcified thoracic aneurysmal wall (Fig. 3A). Flexion–extension films demonstrated considerable movement between the two vertebrae.

CT (Fig. 3B) revealed a lower thoracic aneurysm, 6 cm in diameter, with a contained rupture posteriorly, demonstrating extensive erosion of the adjacent vertebral bodies. The hematoma was surrounded by a thick enhancing wall suggestive of a chronic process.

Urgent surgical repair with spinal stabilization was planned after appropriate medical work-up and orthopedic consultation. While awaiting surgery, however, the patient became hypertensive, had with an increase in back pain and showed evidence of fresh retroperitoneal extension of hematoma on repeat CT.

He underwent emergency aneurysmal repair with a Dacron tube graft without spinal stabilization. The CT findings were confirmed intraoperatively. Gram's staining of the clot revealed 2+ pus but no organisms. Culture grew *Streptococcus faecalis* (2+), *S. epidermidis* (1+) and *E. coli* (1+). Cefazolin was given perioperatively for four doses only.

The patient required repeat exploration for intercostal bleeding and also required prolonged postoperative ventilatory support. He refused subsequent spinal stabilization. He was discharged from hospital 20 days after admission in satisfactory condition. CT 18 months postoperatively showed an organizing paragraft hematoma with calcification but no evidence of infection. At 2 years' follow-up he continues to do well, although he suffers from persistent lower back pain.

Discussion

Rupture of an abdominal aortic aneurysm may be readily diagnosed when the symptomatic triad of ab-

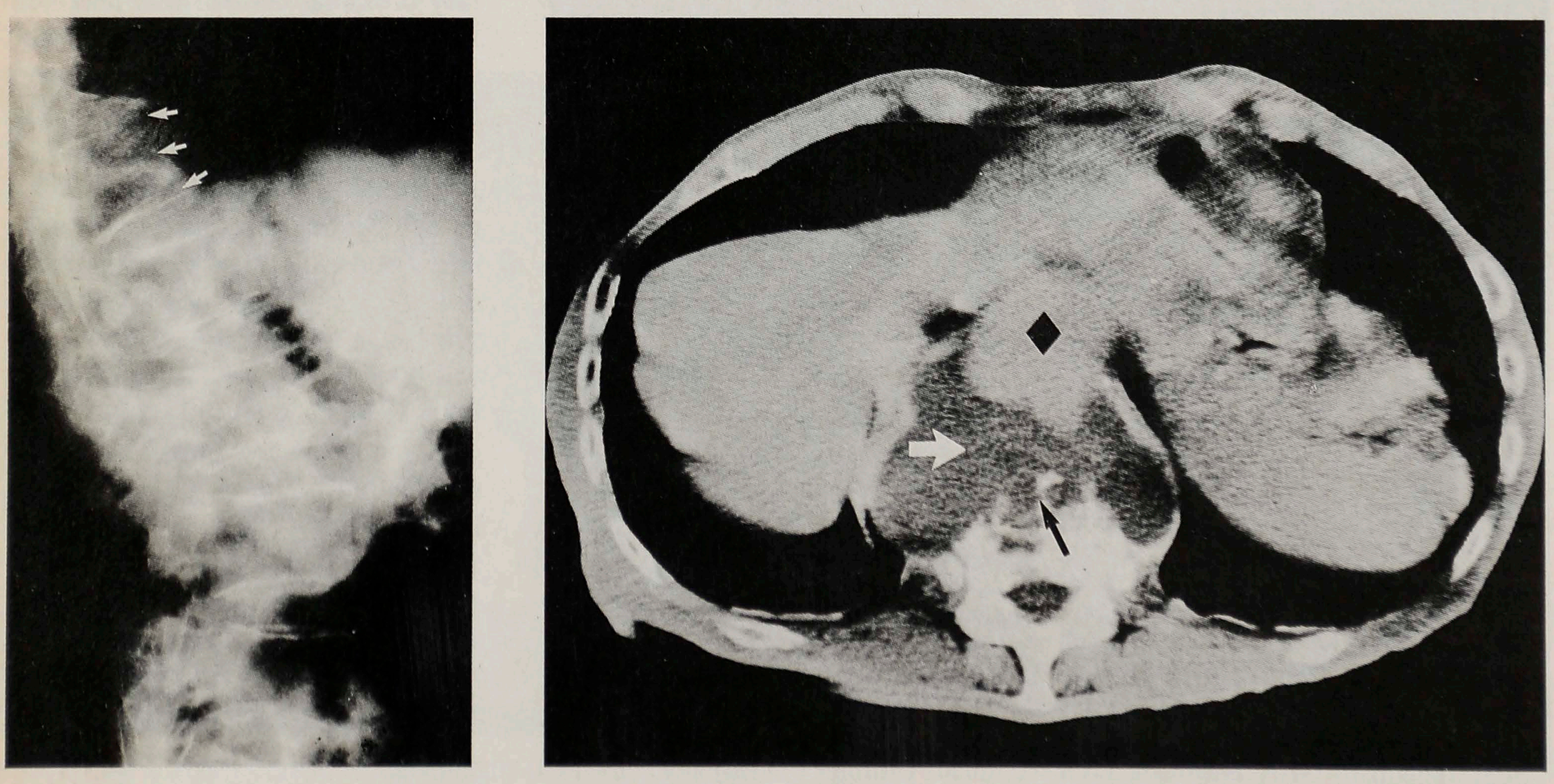

FIG. 3. Case 3. (Left) Lateral x-ray film of thoracolumbar spine shows destruction and collapse of T11 and T12 vertebral bodies with obliteration of intervertebral spaces (arrows). (Right) CT scan demonstrates thoracic aortic aneurysm (diamond), large contained retrocrural hematoma (white arrow) and extensive vertebral body erosion (black arrow).

dominal pain, hypotension and a pulsatile abdominal mass is present. In rare cases, however, the rupture with subsequent hematoma is effectively contained by the surrounding retroperitoneal tissues and bony structures. Under these circumstances the presentation is often atypical and the diagnosis commonly delayed.

Reports of this entity in the literature suggest that chronically contained ruptured abdominal aortic aneurysms make up about 4% of all ruptured abdominal aortic aneurysms, with a range from 2% to 30%.[1] The prohibitive risk of uncontrolled rupture of a previously contained aortic rupture with its incumbent high mortality has been documented by several investigators.[1–5] Urgent repair, on the other hand, has resulted in morbidity and mortality approaching that of elective aneurysm repair.[1,6]

A contained rupture may develop as a result of a relatively small leak associated with minor hemorrhage. The other contributing factor to the genesis of contained rupture is the high resistance of the surrounding structures to extravasation. Most contained ruptures perforate posteriorly,[1–3,5–8] just adjacent to the vertebral body. This strong structure tamponades the rupture, as do the psoas muscle and anterior and posterior renal fascia.[2,6,9] This helps to explain why a significant percentage of contained ruptures have associated vertebral erosion. Whereas about 2% of uncontained ruptures have associated vertebral body erosion, up to 25% of contained ruptures demonstrate this finding,[3,6,10] which may be the result of the combined effect of blood in direct contact with bone and repetitive compression by the pulsatile aneurysm. The effect was dramatic in two of our cases. Vertebral erosion has been a contributing factor to misdiagnosis in several reported cases.[3,5,10,11]

A number of publications have reported unusual presentations of this entity. Szilagyi, Elliott and Smith[4] described five cases simulating sepsis. Others have described uncommon presentations due to the effects of the retroperitoneal hematoma. Some of these include cases causing obstructive jaundice[12,13] simulating a symptomatic inguinal hernia[14] and presenting with femoral neuropathy.[8,15] Still others have described initial misdiagnoses that were attributable to erosion into underlying vertebrae. One case simulated pyogenic vertebral spondylitis,[11] and another imitated metastatic carcinoma.[5]

Our second case simulated sepsis, whereas in the first case the patient presented with an ischemic foot, the initial symptoms of which coincided with an episode of severe back pain. This occurrence likely represented embolization at the time of initial rupture and is the first such reported case.

The third case, although representing a chronically contained thoracic aneurysm, followed many of the precepts of its abdominal counterpart. The initial investigations were undertaken to rule out malignant disease.

All three patients had a delay in diagnosis and associated vertebral erosion. Definitive diagnosis was made by CT. All patients underwent urgent operative repair, and two patients demonstrated evidence of acute hemorrhage preoperatively, underlining the gravity of this diagnosis. All three patients survived surgery.

Chronically contained rupture of aortic aneurysms represents an important subset of ruptured aortic aneurysms and should be considered in elderly people with a history of chronic back pain even in the presence of degenerative disease of the spine, since this could represent vertebral erosion secondary to the ruptured aneurysm.

Hypertension has generally been considered a risk factor for aneurysm rupture. Szilagyi, Elliott and Smith[16] found that about 67% of patients with ruptured aneurysms were hypertensive compared with only 23% of patients with intact aneurysms. The majority of those with contained rupture are not hypertensive.[1,3,6,15] All our patients were normotensive.

Organisms have been cultured in 15% of abdominal aortic aneurysms.[17] Organisms were cultured from the aneurysm contents in all our patients, suggesting that low-grade infection may contribute to this type of rupture. This feature has not previously been identified as a causative factor in contained rupture of aortic aneurysms. Although we do not usually recommend long-term administration of antibiotics in patients with aneurysms, because of the destructive nature of these aneurysms it was thought desirable to add antibiotics to the management protocol.

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Definition and Management of Abdominal Aortic Aneurysms: Results From a Canadian Survey

David Moher, MSc;* C. William Cole, MD, FRCSC;† Gerry B. Hill, MB, MS, FRCP*†

Objective: To assess how vascular surgeons define and manage asymptomatic abdominal aortic aneurysms (AAAs) 5.4 cm in diameter or smaller.
Design: Descriptive survey.
Setting: All provinces and territories of Canada.
Participants: One hundred and ninety-one vascular surgeons.
Interventions: A 12-item questionnaire sent in two mailings to all vascular surgeons who were members of the Canadian Society for Vascular Surgery.
Main Outcome Measures: Definition and routine management of asymptomatic AAAs, assessment of aneurysm size before elective surgery and basic personal data.
Results: One hundred and sixty-seven (87%) vascular surgeons responded to the mailings; 149 (78%) completed the questionnaire. The majority of respondents were experienced (78%), academic (70%) vascular surgeons. AAAs were defined in any one of four ways. There was notable consistency regarding the size range in which vascular surgeons seriously considered elective surgical repair of small aneurysms.
Conclusion: Standards need to be developed to define what constitutes a small AAA if meaningful research on small aneurysms is to be conducted.

Objectif : Évaluer comment les chirurgiens vasculaires définissent et traitent les anévrismes aortiques abdominaux asymptomatiques (AAA), 5,4 cm de diamètre ou moins.
Conception : Une enquête descriptive.
Contexte : Toutes les provinces et tous les territoires du Canada.
Participants : Cent quatre-vingt-onze chirurgiens vasculaires.
Interventions : Un questionnaire en 12 points fut posté en deux envois à tous les chirurgiens vasculaires, membres de la Société canadienne de chirurgie vasculaire.
Principaux effets mesurés : La définition et le traitement courant des AAA asymptomatiques, l'évaluation de la taille des anévrismes avant une chirurgie non urgente et des données personnelles de base.
Résultats : Cent soixante-sept chirurgiens (87 %) ont répondu aux envois postaux; 149 (78 %) ont complété le questionnaire. la majorité de ceux qui ont répondu étaient des chirurgiens vasculaires expérimentés (78 %), du milieu académique (70 %). Les AAA furent définis de quatre façons. On a pu remarquer une uniformité remarquable quant à la taille pour laquelle les chirurgiens vasculaires envisagent sérieusement une intervention non urgente pour réparer de petits anévrismes.
Conclusion : Il y a lieu de développer des normes pour définir ce qui constitue un petit AAA si l'on veut que la recherche sur les petits anévrismes soit significative.

*From the *Clinical Epidemiology Unit, Loeb Medical Research Institute, Ottawa Civic Hospital and the †Division of Vascular Surgery, University of Ottawa, Ottawa, Ont.*

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Reprint requests to: David Moher, MSc, Clinical Epidemiology Unit, Loeb Medical Research Institute, Ottawa Civic Hospital, 1053 Carling Ave., Ottawa, ON K1Y 4E9

An important reason for conducting randomized controlled trials (RCTs) is that their results may lead to changes in clinical practice. Collins and Julian[1] demonstrated that the results of the International Study for Infarct Survival (ISIS) led to a marked increase in the routine use of antiplatelet therapy for treatment of myocardial infarction.

Two RCTs (the U.K. Small Aneurysm Trial and the Veterans' Administration Aneurysm Trial) are currently evaluating whether elective surgical repair or a watch-and-wait approach for asymptomatic abdominal aortic aneurysms (AAAs) ranging in size from 4.0 to 5.4 cm in diameter reduces mortality.[2] When the results of these trials become available it will be important to document any changes in the management of patients with AAA. The first step is to determine the current management of patients with small (4.0 to 5.4 cm) asymptomatic AAAs.

In this paper we present the results of a national survey to determine how Canadian vascular surgeons define and manage asymptomatic AAAs.

Method

We developed a 12-item questionnaire, which addressed three important content areas regarding the definition and management of asymptomatic AAAs. The questions elicited the following information: how vascular surgeons define (according to four published definitions [Table I[3–6]]) and routinely manage AAAs, how they assess the size of an AAA before elective surgical repair, and basic personal information. The survey took less than 10 minutes to complete.

The Canadian Society for Vascular Surgery (CSVS) gave us a list of its current members. The survey was mailed to all of the names on the list at the beginning of November 1991; a second mailing was sent approximately 2 months later to those who had not responded.

The data were tabulated as frequencies and percentages. Differences between geographic regions were assessed with contingency tables.[7] Probability values of less than 5% were considered statistically significant.

Results

Of 191 questionnaires mailed, 167 were returned. Eighteen of the 167 respondents did not complete the questionnaire for the following reasons: 3 did not agree with the questionnaire, 4 had retired and 11 had recently moved. There were 149 questionnaires available for study, a 78% response rate.

For ease of presentation, Canada was divided into four geographic regions; East (Newfoundland, Prince Edward Island, Nova Scotia and New Brunswick), Quebec, Ontario and the West (Manitoba, Saskatchewan, Alberta and British Columbia). The majority of respondents were experienced (78%), academic (70%) vascular surgeons living in Canada (93%). The remaining surgeons (7%) were from the United Kingdom or the United States (Table II). The largest number of vascular surgeons had their surgical practice in the province of Ontario (41%).

The definition of AAAs suggested by the International Society for Cardiovascular Surgery (ISCVS) and the Society for Vascular Surgery (SVS) was most frequently cited (46%) (Table III). There were statistically significant regional differences in how vascular surgeons defined an AAA (ISCVS/SVS definition versus other definitions) ($\chi^2 = 6.14$, df = 3, $p = 0.055$): the SVS definition was more accepted in Ontario and in the West.

The majority (67%) of vascular surgeons routinely assessed AAAs too small to be considered for elec-

Table I. Definitions of Abdominal Aortic Aneurysm Provided in the Survey

Series	Definition
McGregor et al, 1975[3]	The infrarenal aortic diameter must be at least 3.0 cm
Sterpetti et al, 1987[4]	The infrarenal aortic diameter must be at least 1.5 times the suprarenal diameter
Collin, 1990[5]	The maximum infrarenal aortic diameter must be at least 4.0 cm or exceed the maximum diameter of the aorta between the origin of the superior mesenteric and left renal arteries by at least 0.5 cm
International Society for Cardiovascular Surgery/ Society for Vascular Surgery (ISCVS/SVS), Johnston et al, 1991[6]	The aortic artery must be at least 50% larger than that of a "normal" aortic artery

Table II. Survey Responses According to Geographic Region

Region	No. (%) of responses
East*	19 (13)
Quebec	26 (17)
Ontario	61 (41)
West†	33 (22)
United Kingdom and United States	10 (7)
Total	149 (100)

*Newfoundland, Prince Edward Island, Nova Scotia, New Brunswick
†Manitoba, Saskatchewan, Alberta, British Columbia

Table III. Current Definition of an Abdominal Aortic Aneurysm

Series definition	No. (%) of respondents
McGregor et al[3]	31 (21)
Sterpetti et al[4]	24 (16)
Collin[5]	14 (9)
ISCVS/SVS[6]	68 (46)
Other	12 (8)

tive surgical repair, once every 6 months (Table IV). There were also statistically significant regional differences in the frequency (every 6 months versus all other times) with which these assessments were carried out ($\chi^2 = 10.37$, df = 3, $p < 0.01$): surgeons in the East and Quebec were more likely to assess the aneurysm's size every 6 months. For routine assessment of the aneurysm's size all surgeons used ultrasonography; a small proportion (17%) also used computed tomography (CT).

The majority (82%) of vascular surgeons responded that they seriously consider elective surgical repair when the patient's aneurysm is between 4.5 and 5.4 cm in diameter (Table V). This finding was consistent across all geographic regions ($\chi^2 = 2.14$, df = 3, $p > 0.5$). When considering elective surgical repair, almost all (97%) vascular surgeons in all geographic regions responded that they verify the size of the aneurysm once or twice. Of those surgeons who reported using two types of verification (46%) of aneurysm size before operation, slightly more than half (53%) reported using ultrasonography and CT, and 80% reported using both anteroposterior and transverse measurements.

Table IV. Frequency of Assessment of Asymptomatic Abdominal Aortic Aneurysms Too Small for Elective Surgical Repair

Frequency interval, mo	No. (%) of respondents
3	14 (9)
6	100 (67)
Other	35 (23)

Table V. Size Range in Which Surgeons Seriously Considered Elective Surgical Repair of Abdominal Aortic Aneurysms

Size, cm diameter	No. (%) of respondents ($n = 145$)
< 4	3 (2)
4 – 4.4	17 (12)
4.5 – 4.9	48 (33)
5.0 – 5.4	74 (51)
> 5.4	3 (2)

Discussion

The high response rate to this survey suggests that the results are widely applicable. There was no general agreement as to how to define an AAA. The respondents used one of four published definitions, and there were significant geographic regional differences in how an AAA was defined. Thus, part of any reported increase in the incidence of this disease may be explained by differences in how it is defined. If agreement cannot be reached as to what constitutes an aneurysm, further research at the low end of the size spectrum will be difficult. For example, it has been suggested that RCTs evaluating the efficacy of propranolol in reducing the expansion of small (less than 4.5 cm) aneurysms should be undertaken.[8] Without agreement as to when a dilated aorta becomes an aneurysm the results from clinical trials will be difficult to interpret. It will also be more difficult to conduct meta-analysis of trials that evaluate interventions at the low end of the size spectrum, if individual trials use different criteria to define an aneurysm.[9]

There are additional consequences of not using a standard AAA definition. We may be reporting differences in the epidemiology of AAAs because of differences in how they have been defined. For example, we have recently shown that the AAA prevalence ratio changes in both magnitude and direction, depending upon the AAA definition used.[10] This means that if one definition is used, specific groups will be considered at higher risk of having an asymptomatic AAA, but with a different definition the same group will be at a lower risk or protected from having an AAA.

The majority of vascular surgeons agreed on the size range (4.5 to 5.4 cm) at which they seriously consider elective surgical repair of an asymptomatic AAA. This finding was consistent across geographic regions. Both the United Kingdom and Veterans' Administration aneurysm trials are randomizing patients whose AAAs measure between 4.0 and 5.4 cm in diameter. The results of this survey suggest that the results of both these trials in the 4.0 to 4.4-cm size window will have minimal applicability to the members of the CSVS.

Significant differences were noted in how often vascular surgeons routinely assess the size of aneurysms too small to be considered for elective surgical repair. If surgeons assess aneurysm size every 6 months it is important that any changes in size observed are real and not due to measurement error.[11] The results of this survey revealed that all vascular surgeons assessed the size of these aneurysms by ultrasonography. The evidence to date, although limited, suggests that there is minimal measurement error when ultrasonography is used to assess the size of an aneurysm.[12,13] However, more research is required in this area.

We found that slightly more than half (53%) of surgeons verify the aneurysm's size twice before operation by two different modalities. This is an important finding, given that the data evaluating the reliability of aneurysm size measurement with two different radiologic modalities are limited.[14]

From this survey, we have obtained important baseline information on how vascular surgeons define and routinely manage asymptomatic AAAs. Once the re-

sults of the two trials currently under way are published, a follow-up survey will be carried out to document any influence of the trials in the routine management of small asymptomatic AAAs.

We thank Dr. Kenneth A. Harris, in his capacity as Secretary of the Canadian Society for Vascular Surgery, for support during this project.

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Use of Desmopressin Acetate to Reduce Blood Transfusion Requirements During Cardiac Surgery in Patients With Acetylsalicylic-Acid-Induced Platelet Dysfunction

David P. Sheridan, MD, FRCPC;*‡ Robert T. Card, MD, FRCPC;* Jaime C. Pinilla, MD;*
Sheila M. Rutledge Harding, MD, FRCPC;*‡ Dorothy J. Thomson, MD, FRCSC;† Linda Gauthier;* Dawn Drotar*

Objective: To determine whether desmopressin acetate (DDAVP) has the ability to reduce blood loss in patients with a known bleeding tendency.
Design: A randomized, double-blind, placebo controlled study.
Setting: A university teaching hospital.
Patients: Men under the age of 70 years who had taken acetylsalicylic acid within 7 days of scheduled coronary artery bypass surgery. Patients with an abnormal hematologic profile or a history of bleeding or who were receiving heparin or undergoing repeat coronary bypass surgery were excluded. Forty-four patients were randomized with restriction in blocks of 10; 20 received DDAVP and 24 received a placebo.
Main Outcome Measures: Blood loss and blood transfusion requirements.
Results: Patients treated with DDAVP lost significantly ($p < 0.01$) less blood than those receiving a placebo (1543 mL versus 2376 mL respectively). Nineteen patients had a blood loss of more than 2000 mL; 15 of these were in the placebo group. Significantly ($p < 0.02$) fewer patients receiving DDAVP required blood transfusion (9 versus 18).
Conclusions: DDAVP reduces blood loss during cardiac bypass surgery in patients who have taken acetylsalicylic acid within 7 days before operation.

Objectif : Établir si l'acétate de desmopressine peut diminuer la déplétion sanguine chez les malades ayant tendance à saigner.
Conception : Une étude à double insu, randomisée, contre placebo.
Contexte : Un hôpital d'enseignement universitaire.
Patients : Des hommes de moins de 70 ans qui avaient reçu de l'acide acétylsalicylique dans les 7 jours précédant une chirurgie non urgente pour pontage aortocoronarien. Les patients possédant un profil hématologique anormal ou ayants des antécédents de saignement, ou qui recevaient de l'héparine ou qui en étaient à un second pontage furent exclus. Quarante-quatre patients furent randomisés par blocs de 10; 20 reçurent de la desmopressine et 24, un placebo.
Principaux effets mesurés : Les pertes sanguines et les transfusions sanguines requises.
Résultats : Les patients traités à l'acétate de desmopressine ont perdu significativement moins de sang ($p < 0,01$) que ceux qui recevaient un placebo (1543 mL contre 2376 mL, respectivement). Dix-neuf patients subirent des pertes sanguines dépassant 2000 mL; 15 d'entre eux étaient dans le groupe placebo.

*From the *Department of Medicine, †Department of Surgery and ‡Department of Pathology, University of Saskatchewan, Saskatoon, Sask.*

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Reprint requests to: Dr. David P. Sheridan, Division of Hematology, Department of Medicine, Royal University Hospital, University of Saskatchewan, Saskatoon, SK S7N 0X0

Significativement moins de patient recevant de l'acétate de desmopressine ($p < 0,02$) nécessitèrent une transfusion (9 contre 18).
Conclusions : L'acétate de desmopressine diminue la déplétion sanguine en cours de pontage aortocoronarien chez les patients qui ont pris de l'acide acétylsalicylique dans les 7 jours qui précèdent l'opération.

The most common and clinically relevant change in the hemostatic mechanism during cardiopulmonary bypass is a temporary loss of platelet function.[1] This hemostatic dysfunction is aggravated in patients who have been taking acetylsalicylic acid (ASA) preoperatively. Such patients have increases in postoperative blood loss, transfusion requirements and incidence of reoperation for hemorrhage.[2–4] Higher transfusion requirements in turn expose patients to increased risks related to blood transfusion.

Simple withdrawal of ASA, however, may not be the ideal solution to this problem, because many candidates for cardiac arterial bypass grafting (CABG) have unstable angina: in a review of the literature on the use of ASA in ischemic heart disease, Willard, Lange and Hillis[5] concluded that patients with unstable angina have a 50% to 70% reduction of myocardial infarction and death with ASA use. The Steering Committee of the Physicians' Health Study Research Group showed conclusively that ASA reduced the risk of myocardial infarction.[6] The UK-TIA study group[7] also demonstrated that in patients taking ASA there were fewer nonfatal strokes and nonfatal myocardial infarctions than among control patients. Furthermore, it has been shown that patients who take ASA in the perioperative period have a reduced incidence of graft failure.[5] It may be advantageous, therefore, for patients who are scheduled to undergo CABG to take ASA both preoperatively and postoperatively.

Intraoperative and postoperative blood loss is a serious problem associated with cardiopulmonary bypass surgery. Interventions to reduce bleeding are often evaluated but few have proven to be of benefit.

Desmopressin acetate (DDAVP) has been found to shorten the bleeding time,[8] thus reducing blood loss. It acts by inducing the release of von Willebrand factor from the endothelial cells.[9] DDAVP (1-desamino-8-D-arginine vasopressin) is a synthetic analogue of vasopressin but lacks its vasoconstrictor properties. It has been postulated that DDAVP would reduce blood loss and subsequent transfusion requirements in patients who undergo cardiac surgery, but the evidence for this is controversial. Three studies did not demonstrate a significant difference in blood loss or in blood transfusion requirements between control patients and DDAVP-treated patients during cardiac surgery.[10–12] However, only one of these studies[10] included patients who took ASA preoperatively. Two studies have shown reduced bleeding after treatment with DDAVP. In the first study, by Rocha and colleagues,[13] total blood loss was not reduced, but DDAVP was effective in reducing intraoperative bleeding in patients placed on cardiopulmonary bypass. In the other study, by Salzman and colleagues,[14] DDAVP significantly reduced mean operative and early postoperative blood loss in patients who underwent various cardiac operations requiring cardiopulmonary bypass. Neither of these two studies reported on ASA use.

Because of these conflicting findings, we speculated that the benefits of DDAVP may only be seen in patients at increased risk of bleeding, including patients who continue to take ASA up to the time of surgery. We therefore conducted a prospective, randomized, double-blind trial on patients who were taking ASA within 7 days before CABG.

Patients and Methods

The 44 patients enrolled in the study were men under the age of 70 years who had taken ASA within 7 days of scheduled bypass surgery. Patients with an abnormal hematologic profile (hemoglobin concentration, platelet count and prothrombin or partial thromboplastin times or a history of bleeding) were excluded, as were patients receiving heparin or those scheduled for repeat bypass surgery. Informed consent was obtained at the time of enrolment. The study was approved by the University of Saskatchewan Ethics Committee. Randomization was done with restriction, in blocks of 10.

Twenty patients randomized to the treatment group received 10 μg/m^2 body surface area of DDAVP diluted in saline, infused intravenously over 20 minutes after completion of cardiopulmonary bypass and reversal of the heparinization. Twenty-four patients randomized to the control group were infused with 20 mL of saline instead of the DDAVP but were otherwise treated identically.

Blood loss was evaluated throughout the operative and postoperative periods. Loss was measured in sponges (by weighing them), suction, drainage and cardiopulmonary bypass pump residual. Hemoglobin concentration, platelet count and prothrombin and partial thromboplastin times were measured preoperatively, intraoper-

atively and 1 hour, 4 hours and 24 hours postoperatively.

Results are presented as means with 95% confidence intervals (CI). Student's *t*-test was used to compare mean values. The consistency of proportional data was determined by the χ^2 test.

Results

Both groups were similar in regard to age, number of bypasses performed, preoperative hemoglobin concentration, platelet count, prothrombin and partial thromboplastin times (Table I). One patient in the control group and one patient in the DDAVP group suffered a cerebrovascular accident perioperatively. There were no perioperative deaths in either group.

The only difference in laboratory values between the two groups was in the partial thromboplastin time at 1 hour postoperatively. It was significantly ($p < 0.001$) shorter in the DDAVP-treated group than in the control group (34 seconds, 95% CI 32.3 to 35.7 seconds versus 40 seconds, 95% CI 37.1 to 43.1 seconds). By 4 hours postoperatively, the times were again similar.

In all, there were 19 patients who lost more than 2000 mL of blood; the majority (15 patients) were in the control group ($p < 0.01$). Nineteen of the 24 control patients received a blood product perioperatively compared with only 9 of the 20 treated patients ($p < 0.02$). Eighteen control patients received concentrated red cells, 8 received platelets, and 12 received plasma. Nine treated patients were administered concentrated red cells, three received platelets, and six received plasma. The amount of blood products used was less for those treated with DDAVP than for the controls, although the difference did not reach statistical significance.

The group of patients treated with DDAVP had significantly ($p < 0.01$) decreased total blood loss when compared with that of the control group (1543 mL, 95% CI 1269 to 1817 mL versus 2376 mL, 95% CI 1859 to 2893 mL). For comparison, the blood loss in 50 patients not taking ASA who underwent bypass surgery in our hospital was evaluated retrospectively. Their mean blood loss was 1359 mL, considerably less than the 2376 mL in the ASA control group. This finding confirmed that patients treated with ASA have increased bleeding during cardiac surgery. This tendency was not completely corrected by treatment with DDAVP, although the blood loss in the DDAVP treated patients was similar to the non-ASA-treated, retrospective controls.

Table I. Characteristics of Patients With Acetylsalicylic-Induced Platelet Dysfunction Who Received or Did Not Receive Desmopressin Acetate (DDAVP) During Cardiac Surgery

Characteristic	DDAVP, mean* (range)	Control, mean* (range)
No. of patients	20	24
Age, yr	56.6 (52.9 – 60.3)	61.6 (58.6 – 64.6)
No. of bypasses	3.1 (2.6 – 3.6)	3.5 (3.1 – 3.9)
Hemoglobin level, g/L	148 (142 – 154)	148 (144 – 152)
Platelet count, X 10^9/L	270 (241 – 301)	270 (252 – 289)
Prothrombin time, s	11.9 (11.6 – 12.2)	11.7 (11.5 – 11.9)
Partial thromboplastin time, s	29.6 (27.9 – 31.3)	29.2 (27.7 – 30.9)

*95% confidence intervals

Discussion

Previous studies of DDAVP in patients who undergo cardiac surgery have given conflicting results.[9–13] Our study demonstrated reduced blood loss and transfusion requirements in patients with a known bleeding tendency (ASA-induced platelet dysfunction) who underwent cardiopulmonary bypass. Previous studies of DDAVP in cardiac surgery that have evaluated bleeding and transfusion requirements have largely involved patients without a bleeding tendency. Many of these patients have minimal blood loss, so it is not surprising that an intervention to reduce bleeding did not result in significant benefit.

Our data demonstrate that a majority of the patients who were taking ASA preoperatively could be operated on without any requirement for blood transfusion. This represents a major clinical benefit for patients who remain on their ASA up to the time of their cardiac surgery. Without DDAVP, the majority of patients who undergo cardiac surgery will require either that ASA be discontinued before surgery or that blood product replacement be given during the perioperative period.

Conclusion

Use of DDAVP is recommended during cardiac surgery for patients in whom discontinuation of ASA therapy poses a significant risk.

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BOOKS RECEIVED
LIVRES REÇUS

This list is an acknowledgement of books received. It does not preclude review at a later date.

Cette liste énumère les livres reçus. Elle n'en exclut pas la critique à une date ultérieure.

Acute Pancreatitis: Diagnosis and Therapy. Edited by Edward L. Bradley III. 294 pp. Illust. Raven Press, Ltd., New York. 1993. $125 (US). ISBN 0-7817-0091-4

Atlas of Urosurgical Anatomy. Frank Hinman, Jr. 553 pp. Illust. W.B. Saunders Company/Harcourt Brace Jovanovich, Inc., Philadelphia. 1993. $183. ISBN 0-7216-3955-0

Biological, Material, and Mechanical Considerations of Joint Replacement. Edited by Bernard F. Morrey. Bristol-Myers Squibb/Zimmer Orthopaedic Symposium Series. 470 pp. Illust. Raven Press, Ltd., New York. 1993. $98 (US). ISBN 0-7817-0008-6

Clinical Atlas of Transesophageal Echocardiography. Martin E. Goldman; with contributions by Bruce P. Mindich, Allen Mogtader and Theresa Guarino. 376 pp. Illust. Futura Publishing Company Inc., Mount Kisco, NY. 1993. $149 (US). ISBN 0-87993-539-1

Conduits for Myocardial Revascularization. Edited by Michel Carrier and L. Conrad Pelletier. 106 pp. Illust. R.G. Landes Company, Boca Raton, Fla. 1993. $89.95 (US). ISBN 1-879702-66-5

Intestinal Stomas: Principles, Techniques and Management. Edited by John M. MacKeigen and Peter A. Cataldo. 414 pp. Illust. Quality Medical Publishing Inc., St. Louis. 1993. $85 (US). ISBN 0-942219-40-6

Laparoscopic Urologic Surgery. Edited by Leonard G. Gomella, Mike Kozminski and Howard N. Winfield. 286 pp. Illust. Raven Press, Ltd., New York. 1993. $120 (US). ISBN 0-7817-0044-2

Peritonitis. Edited by Donald E. Fry. 327 pp. Illust. Futura Publishing Company Inc., Mount Kisco, NY. 1993. $70 (US). ISBN 0-87993-551-0

The Practice of Operational Research. Edited by George Mitchell. 235 pp. John Wiley & Sons, Chichester, UK. 1993. $59.95. ISBN 0-471-93982-X

Surgery for Stroke. Edited by R.M. Greenhalgh and L.H. Hollier. 420 pp. W.B. Saunders Co., London, UK. 1993. $175. ISBN 0-7020-1759-0

Surgical Infections: Diagnosis and Treatment. Edited by Jonathan L. Meakins. 531 pp. Illust. Scientific American Inc., New York. 1993. $89 (US). ISBN 0-89454-014-9

Heparinized Saline Versus Normal Saline in Maintaining Patency of the Radial Artery Catheter

Mohan Kulkarni, MD, FRCSC;* Colleen Elsner, RN, BAA(N), CCRN;† Doreen Ouellet, RN;‡
Robert Zeldin, MD, FRCSC, FACS§

Objective: To compare the ability of normal versus heparinized saline infusion to maintain patency of the radial artery catheter used for monitoring or multiple blood sampling.
Design: Randomized double-blind trial.
Setting: A university-affiliated hospital.
Patients: All 108 patients admitted to the surgical intensive care unit who required radial arterial line catheters. Excluded were patients who required emergency admission, those who refused to give consent, those who needed anticoagulants, thrombolytic or platelet therapy, those whose cannulation site was other than the radial artery, inadvertent discontinuation of the arterial line or incomplete data collection. This resulted in a sample of 78 adults, randomized according to date of admission.
Interventions: Forty patients admitted on odd-numbered dates received heparinized normal saline (2 units/mL), and 38 patients admitted on even dates received normal saline, both as continuous flush solutions.
Main Outcome Measures: Measurement of radial artery flow and pressure before and after cannulation, and catheter patency during cannulation.
Results: The type of flush solution did not adversely affect the radial artery or the hand in any measurable way. Catheter blockage occurred in three patients receiving heparinized saline and seven patients receiving normal saline as the flush solution ($p = 0.06$). At 96 hours of cannulation, 92% of the catheters in the heparinized saline group were patent compared with 74% in the normal saline group. Intra-arterial blood pressure was inaccurate compared with the brachial cuff pressure in 6 patients in the heparinized saline group compared with 14 patients in the normal saline group ($p < 0.03$).
Conclusions: There is no significant difference between flushing with normal saline and heparinized saline in the maintenance of radial arterial line patency. However, the use of a continuous heparinized flush solution in pressurized arterial lines is beneficial in that it results in greater accuracy of blood pressure monitoring than normal saline infusion.

From the Surgical Intensive Care Unit, Toronto East General & Orthopaedic Hospital Inc., Toronto, Ont.

Supported by a grant from The Research Foundation of Toronto East General & Orthopaedic Hospital Inc.

**Fellow, Department of Surgery, Toronto East General & Orthopaedic Hospital Inc.*

†*Nurse educator, Surgical Intensive Care Unit and Post-Anesthetic Recovery Room, Toronto East General & Orthopaedic Hospital Inc.*

‡*Nursing Unit Manager, Surgical Intensive Care Unit, Toronto East General & Orthopaedic Hospital Inc.*

§*Director, Surgical Intensive Care Unit, Toronto East General & Orthopaedic Hospital Inc., and Department of Surgery, University of Toronto, Toronto, Ont.*

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Reprint requests to: Dr. Robert Zeldin, Director, Surgical Intensive Care Unit, Toronto East General & Orthopaedic Hospital Inc., 825 Coxwell Ave., Toronto, ON M4C 3E7

Objectif : Comparer la capacité respective d'une perfusion de solution physiologique salée normale et celle d'une solution salée héparinée pour maintenir la perméabilité du cathéter de l'artère radiale utilisé pour fins de monitoring ou pour effectuer des prélèvements sanguins multiples.
Conception : Une étude randomisée à double insu.
Contexte : Un hôpital universitaire.
Patients : Cent huit patients reçus à l'unité de soins chirurgicaux intensifs et qui avaient besoin d'un cathétérisme de l'artère radiale. Furent exclus les patients nécessitant une hospitalisation en urgence, ceux qui ont refusé leur consentement, ceux qui avaient besoin d'anticoagulants, d'un traitement thrombolytique ou de plaquettes, ceux chez qui un cathéter avait été posé ailleurs que dans l'artère radiale, les interruptions imprévues de la perfusion artérielle ou lorsque les données étaient incomplètes. Ceci a laissé un échantillonnage de 78 adultes, randomisés selon leur date d'hospitalisation, qui ont reçu soit une solution salée normale, soit une solution salée héparinée comme perfusion de rinçage.
Interventions : Quarante patients hospitalisés lors de dates impaires ont reçu une solution salée héparinée (2 unités/mL) et 38 patients reçus aux jours pairs ont reçu une solution salée normale. Dans les deux cas, les solutions ont été administrées commme perfusion de rinçage.
Principaux effets mesurés : Le débit de l'artère radiale et la pression avant et après la canulation, et la perméabilité du cathéter durant la canulation.
Résultats : Le type de solution de rinçage n'a pas montré d'effet indésirable sur l'artère radiale ni sur la main, d'aucune façon mesurable. Un blocage du cathéter est survenu chez trois patients qui recevaient la solution héparinée et chez sept patients qui recevaient la solution salée normale ($p = 0,06$) comme perfusion de rinçage. Après 96 heures de canulation, 92 % des cathéters du groupe hépariné étaient perméables comparativement à 74 % pour la solution salée normale. Comparativement à la pression mesurée au sphygmomanomètre, la pression intra-artérielle était inexacte chez 6 patients du groupe hépariné par rapport à 14 patients dans l'autre groupe ($p < 0,03$).
Conclusions : Il n'y a aucune différence significative entre un rinçage avec une solution salée héparinée ou avec une solution salée normale en ce qui a trait au maintien de la perméabilité d'un cathéter de l'artère radiale. Toutefois, l'utilisation d'une perfusion continue de solution salée héparinée dans une ligne artérielle sous pression est bénéfique. La perfusion de soluté hépariné permet une plus grande précision du monitoring de la tension artérielle qu'une perfusion de solution salée normale.

Indwelling radial artery catheters are invaluable for providing continuous monitoring of systemic blood pressure, and they are thereby a convenient port for multiple blood sampling. With appropriate indications and careful technique, the complications arising from these catheters can be kept to a minimum.[1–4] Although it is clear that continuous pressurized flushing is better than intermittent manual flushing for maintaining catheter patency,[5,6] the value of heparin in the flush solution remains largely speculative and unproven. Two recent studies that compared the use of heparinized and nonheparinized solutions in radial arterial lines presented conflicting results. Hook and associates[7] concluded that short-term catheter patency could be maintained with the use of a nonheparinized flush solution, whereas Clifton and associates[8] discontinued their study because of a significant difference in patency rates with nonheparinized flush solutions.

Heparin sodium is an anticoagulant that enhances the action of antithrombin III, an inhibitor of thrombin. Heparin also neutralizes the activated forms of coagulation Factors XII, X and possibly VII and platelet factor 1. This may prevent or retard the deposition of fibrin in and around the catheter, thus allowing accurate reading of the intra-arterial blood pressure.

There are many potential side effects of heparin use in radial artery catheters, including abnormal results of coagulation studies, bleeding dyscrasias and thrombocytopenia.[9–11] We designed a study to determine the safety, efficacy and potential cost savings of maintaining patency of radial arterial lines with normal saline as opposed to heparinized normal saline. The clinical end point for comparison was catheter blockage, which was defined as any one of the following occurrences that could not be corrected with catheter manipulation: inability to withdraw blood from the catheter, inability to flush the catheter and a flat intra-arterial pressure waveform. It was hypothesized that patients who received normal saline in continuous pressurized arterial lines would maintain a patency rate equal to those of patients who received heparinized normal saline. To test this hypothesis, we initiated a prospective, randomized double-blind trial in the Surgical Intensive Care Unit at our institution.

Patients and Methods

The study protocol was submitted

for review and approved by the Clinical Development Review Committee at Toronto East General & Orthopaedic Hospital Inc. Seventy-eight adults admitted to the Surgical Intensive Care Unit between May 1, 1990, and Jan. 30, 1991, who required radial arterial monitoring, were entered into the study after informed consent had been obtained. All patients were hemodynamically stable, without any clinically significant peripheral vascular disease. All arterial lines were inserted preoperatively.

The patients were randomly allocated to two groups, according to the date of admission to hospital: group A for patients admitted on odd-numbered dates and group B for those admitted on even-numbered dates. Group A patients (n = 40) received heparin sodium (1000 units/500 mL of 0.9% sodium chloride) as the arterial line flush solution. Group B patients (n = 38) received 0.9% sodium chloride alone as the arterial line flush solution. A continuous pressure of 300 mm Hg was applied with a C-Fusor 500 pressure bag (Medical Medox Inc., Dynatec, Toronto) and a flow rate of approximately 3 mL/h was delivered through a Baxter Pressure Monitoring Kit with Uniflow Disposable Transducer (Baxter Healthcare Corp., Edwards Critical-Care Division, Irvine, Calif.). The flush solutions, labelled A or B, were premixed by the hospital's pharmacy department to facilitate a double-blind study.

Patients were excluded from the study if they required emergency admission or the therapeutic use of anticoagulants, thrombolytic therapy or antiplatelet therapy (with the exception of subcutaneous heparin therapy for prophylaxis of deep venous thrombosis). Patients were also excluded if the arterial line was in a site other than the radial artery, the radial artery could not be cannulated within five attempts, the data were incomplete or consent was not obtained.

Procedure

All patients underwent baseline, daily and post-catheter-removal assessment by selected and specially trained critical care nurses.

The baseline assessment included the following:

- Allen's test for the diagnosis of chronic occlusive arterial lesions distal to the wrist.[12] The results were graded as follows: grade 1, 0 to 7 seconds (normal); grade 2, 8 to 14 seconds (equivocal); grade 3, 15 seconds or longer (poor).[13]
- Measurement of finger pulse waveform recordings on the first, third and fifth digit with the Mennen Finger Pulse Oximetry Probe and Monitor (Mennen Medical Inc., Clarence, NY). A difference of 25% in the height of the waveform was considered significant.[2]
- Measurement of bilateral digit temperature on the first, middle and little digits with the First Temp tympanic thermometer (Critical Assist Group Inc., Markham, Ont.). A change between digits of 2°C between baseline and post-removal temperatures was considered significant.
- Assessment of radial artery flow assessment with a Doppler ultrasonic flowmeter[14] (Narco Scientific, Medasonics, Mountain View, Calif.). The findings were graded as follows: grade 1, unobstructed flow; grade 2, obstructed flow; grade 3, no flow.
- Coagulation assessment. This included measurement of the prothrombin time, partial thromboplastin time and platelet count.

The arterial lines were inserted by surgical residents according to the standard protocol established by the research team. With aseptic technique, the site was anesthetized with 2% lidocaine. The artery was cannulated with a Teflon angiocatheter (20 gauge 2 inch)[4,15] (Deseret Medical, Inc., Becton Dickinson and Co., Sandy, Utah). The number of attempts, the technique — direct versus transfixion insertion — and the use of a guide wire (if any) were recorded.

The daily assessment included examination of the following: site appearance, quality of the arterial pressure waveform (dampened versus normal), accuracy of intra-arterial blood pressure measurement compared with brachial artery cuff pressure measurement, ease of blood withdrawal, ability to flush the line, number of times blood was withdrawn from the system, finger pulse waveform, digit temperature and Doppler ultrasonic flowmeter assessment of the radial artery.

After removal of the radial arterial catheter, the baseline assessment was repeated. The length of time (in hours) that the arterial line was in place and the reason for discontinuation of the line were documented.

The distribution of time until blockage occurred was estimated by the Kaplan–Meier technique[16] for each of the two groups. The Mantel log rank test[17] was used to test the equality of the time to blockage distributions.

χ^2 analysis was used to compare the frequencies of events in each group, specifically regarding the accuracy of intra-arterial pressure measurement compared with brachial cuff pressure measurement.

Fisher's exact test and Yates' correction were used to critically examine the statistical findings in relation to the sample size.

Results

Both groups were comparable in terms of demographic features

(Table I), and the results of coagulation studies in both were within normal limits (prothrombin time, 10 to 13 seconds, partial thromboplastin time, 24 to 38 seconds, platelet count, 150 to 300 $\times$ 10^9/L). The mean number of times the line was accessed was 16 for group A and 11 for group B. In both preinsertion and postremoval testing, the results of Allen's test were rated at grade 1 for patients in both groups. Finger pulse waveforms, digital temperatures and Doppler blood flow in the radial artery were within acceptable ranges before and after catheter removal in both groups.

Catheter patency curves for radial artery catheters flushed with heparinized normal saline and normal saline, when the equality of time to blockage distribution was tested, were not significant ($p = 0.06$, Kaplan–Meier and log rank tests) (Fig. 1).

The mean duration of the arterial catheterization was 98 hours in group A and 80 hours in group B. Because this analysis does not take into account the varying lengths of time in which a blockage could occur, Fisher's exact test was applied to the data ($p = 0.18$). At 72 hours, 92% of the catheters in group A and 84% of the catheters in group B were patent. At 96 hours, 92% of the catheters in group A and 74% of the catheters in group B were patent. Eighteen percent more catheters in group A remained patent than in group B, particularly past 72 hours. There were three blocked catheters in group A and seven in group B. This finding was not significant ($p = 0.05 > p \leq 0.16$, χ^2 test). The confidence intervals for the difference in proportion were −0.643, +0.283, Yates' correction). The intra-arterial blood pressure was found to be inaccurate compared with brachial cuff pressure values in 6 (15%) of 40 catheters in group A and 14 (37%) of 38 catheters in group B ($p < 0.03$, χ^2 test, $p = 0.04$ Fisher's exact test). The confidence intervals for the difference in proportion were +0.433, +0.0035, Yates' correction.

Table I. Demographic Features of 78 Adult Surgical Patients Who Required Insertion of Radial Artery Catheters

Characteristic	Group A*	Group B†
Sex		
Male	29	25
Female	11	13
Age, yr		
Mean	66.7	65.9
Range	21–87	38–86

*Catheter flushed with heparinized saline solution.
†Catheter flushed with normal saline solution.

Discussion

Since the morbibity of radial artery cannulation is less than 1%, a definitive sample size would ideally comprise 168 patients. However, it was not feasible to secure a sample of such a size in this clinical setting, given the limited time frame and resources. Nevertheless, the study yielded the largest sample yet for the study of this subject. Previous reports cited sample sizes of only 30[8] and 50.[7]

In this study, the use of normal saline did not adversely affect arterial flow in the radial artery or the hand. The incidence of hand ischemia has been reported to be as high as 10% with radial artery catheterization[3,18] yet we found no cases of it in either group.

Although two different methods of cannulation were used, transfixion and direct, neither adversely affected the radial artery or the hand. This finding supports that of Jones, Hill and Narwold[19] who were unable to demonstrate an appreciable difference in thrombus formation between these methods of cannulation.

There was no documented change in the results of Allen's test or of the measurement of finger-pulse waveform, digital temperature or the radial artery Doppler pulse blood flow for either group when preinsertion values were compared with postremoval values. These indirect measurements of arterial blood

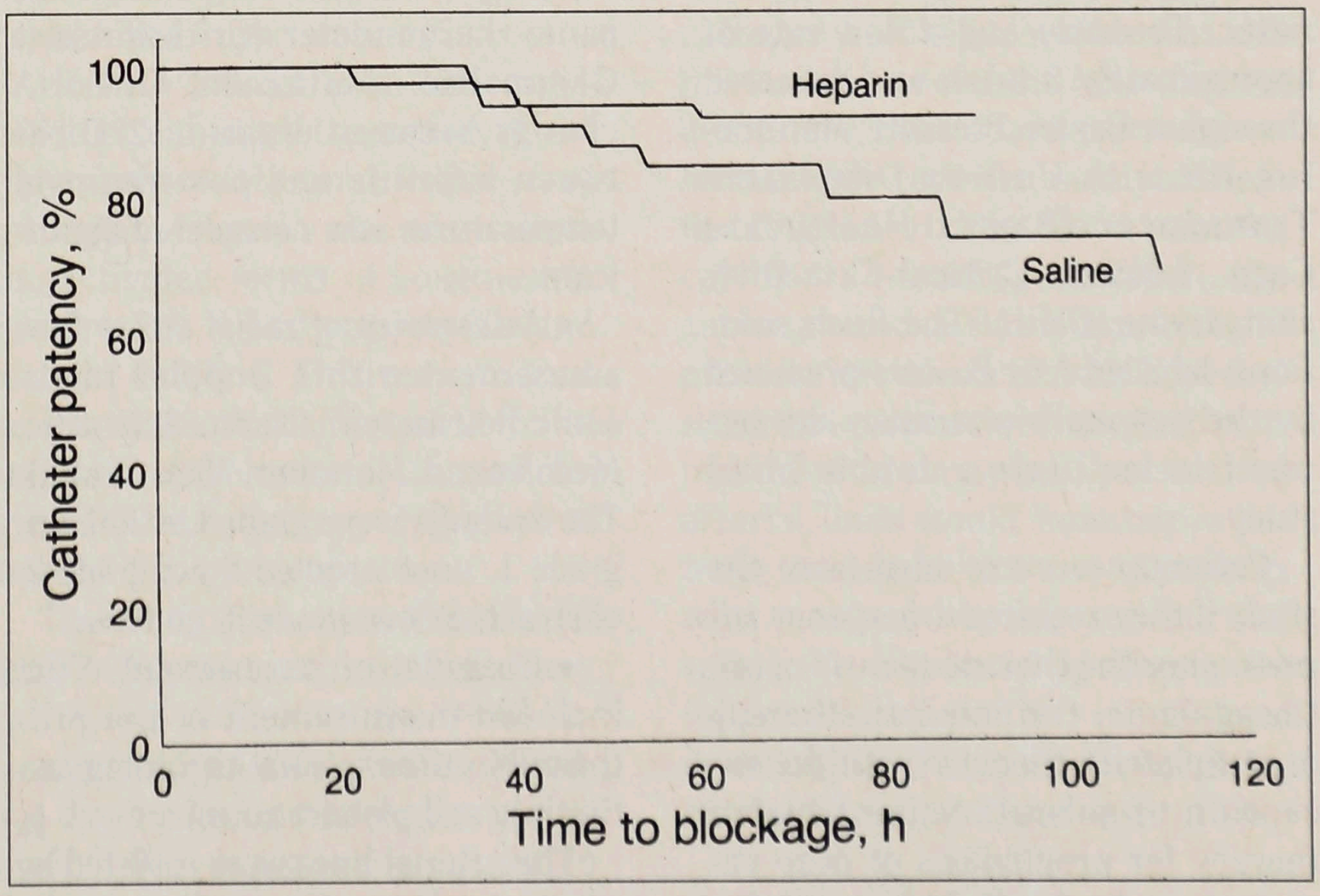

FIG. 1. Patency of radial arterial catheters flushed with heparinized saline solution or normal saline solution. Time to blockage was estimated by Kaplan–Meier technique. Mantel log rank test was used to test equality of time to blockage distributions.

flow suggest that, regardless of the solution used to maintain the patency of arterial lines, arterial perfusion of the limb is not affected. However, adverse affects on arteries have been noted with the use of normal saline flush solution, specifically loss of patency and phlebitis.[20]

Although the number of blocked catheters was higher in the normal saline group (n = 7) than the heparin group (n = 3), this finding was not significant.

In a similar study, Hook and associates[7] concluded that the addition of heparin to the flush solution in arterial lines did not improve patency rates. However, their patients had undergone cardiac surgery and may have received anticoagulation, which is required for cardiopulmonary bypass. Also, their study was neither double-blinded nor randomized.

However, Garrelts, LaRocca and Ast,[21] who studied the effects of heparin versus normal saline flush solutions on indwelling venous catheters, found fibrin strands or thrombi, or both, in malfunctioning catheters flushed with normal saline. Rajani and colleagues[20] found lower rates of occlusion and longer survival of umbilical artery catheters flushed with heparinized saline than those flushed with saline alone.

Although the present study showed no significant difference in patency rates with the two types of flush solution, the addition of heparin seems to have merit in ensuring accurate invasive monitoring of arterial blood pressure.

The measurement of intra-arterial blood pressure was found to be inaccurate compared with brachial cuff blood pressure values in a significant number of patients receiving normal saline flush solution. The results are in agreement with those of Clifton and associates,[8] who found also that the use of saline flush solution in radial artery catheters was associated with an increased frequency of catheter malfunction requiring manipulation of the catheter by the nursing staff. As well, Hook and associates[7] found that the appearance of a good waveform occurred more consistently with heparinized radial artery flush solutions than with nonheparinized solutions.

Although the presence of heparin in the arterial flush solution demonstrates clinical merit, a definitive ratio for heparin in the solution is as yet undetermined. Bolgiano and associates[22] reported acceptable patency rates and accurate waveforms when heparin concentrations of 0.25 to 1 unit/mL were used.

As to heparin sodium's potential side effects, no significant adverse reactions were noted with the use of heparin in the present study.

Conclusions

There is no significant difference between normal saline and heparinized normal saline in maintaining the patency of radial arterial lines. However, the use of a continuous flush solution of heparinized normal saline (2 units/mL) in pressurized arterial lines is beneficial. Use of this solution leads to greater accuracy of intra-arterial blood pressure monitoring than normal saline flush solution. Nevertheless, the optimum concentration of heparin necessary to maintain reliable hemodynamic monitoring warrants further investigation.

Although the difference in the time to catheter blockage was not significant, catheters flushed with heparinized normal saline had a longer duration of patency than those flushed with normal saline. Consideration must be given to the fact that every catheter will eventually become dysfunctional and block. This prevents equitable comparison of the data between the groups. The catheter survival curves suggest that there may be a greater difference in the duration of patency between the groups when the catheters are left in place for greater lengths of time. A larger sample size is required to demonstrate any significant difference in terms of catheter patency rates.

We acknowledge the assistance and commitment of this project's primary data collectors: L. Hamilton, RN, M. Lee, RN, D. Morgan, RN, F. O'Reilly, RN and C. Scherbey, RN. We also thank the staff nurses of the Surgical Intensive Care Unit and the Pharmacy Department of the Toronto East General & Orthopaedic Hospital Inc. for their ongoing support.

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SESAP VII Critique / Critique SESAP VII

Items 82 to 84

Endoscopic retrograde cholangiopancreatography (ERCP) is valuable for patients with chronic abdominal pain and obscure abdominal pain who have previously undergone cholecystectomy. Retained gallstones and pancreatitis will be identified in a substantial number of such patients.

Cholescintigraphy is of major value for diagnosis of acute cholecystitis in a patient whose gallbladder appears normal on ultrasonography despite clinical symptoms of acute cholecystitis. It is also of value for confirming cystic duct obstruction in the poor-risk patient who might require urgent operation.

Ultrasonography has an accuracy of 96% in the diagnosis of gallbladder calculi. It will determine the size of the intrahepatic and extrahepatic bile ducts as well as diagnose gallbladder cancer. It is the preferred initial study for a patient who has jaundice with an apparent obstructive origin.

82 D 83 E 84 B

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Malignant Lymphoma of Bone

Stephen J. Lewis, MD; Robert S. Bell, MD, FRCSC; Bernard J.J. Fernandes, MD, MRCPath, FRCPA, FRCPC; Ronald L. Burkes, MD, FRCPC

Objective: To evaluate the diagnosis and management of patients with malignant lymphoma of bone.
Design: A case series review in which the miniumum follow-up was 24 months and the mean follow-up was 49 months.
Setting: All patients were managed at a tertiary care centre, although initial biopsies were often done in community centres.
Patients: Selected for review were 15 of 18 consecutive patients who were referred to the Musculoskeletal Oncology Unit at the Mount Sinai Hospital, Toronto, between 1984 and 1989, with a bone lesion as the presenting symptom of lymphoma. The three excluded patients included two with diffuse nodal disease at presentation and one who had a second, unrelated malignant tumour.
Interventions: Staging studies (hematologic investigations, radiography, technetium bone scanning and computed tomography), surgical biopsies of the lesion, chemotherapy, radiotherapy and in some cases surgical resection of the lesion.
Main Outcome Measures: The number of biopsies required for diagnosis and the incidence of complications that required operative intervention.
Results: Seven of the 15 patients required more than one biopsy to establish the diagnosis. Five patients required surgical procedures for late complications that included pathologic fractures, wound infection and osteonecrosis. At 24 months' follow-up, 13 patients were disease free and 2 had died.
Conclusions: Proper biopsy and pathological evaluation are crucial in the diagnosis of lymphoma of bone. These measures will decrease the necessity for repeat biopsies. Lymphoma is best managed medically. Surgery should be reserved for biopsy and for treatment of the complications of therapy.

Objectif : Évaluer le diagnostic et le traitement des patients avec lymphomes malins osseux.
Conception : Une étude rétrospective de cas dont le suivi minimum a été de 24 mois et le suivi moyen de 49 mois.
Contexte : Tous les patients furent traités dans un centre de soins tertiaires, bien que la biopsie initiale ait souvent été pratiquée dans des centres communautaires.
Patients : Quinze des 18 patients consécutifs reçus au Service d'oncologie musculosquelettique du Mount Sinai Hospital de Toronto, entre 1984 et 1989, souffrant d'une lésion osseuse comme première manifestation d'un lymphome furent choisis pour l'étude. Les trois patients exclus comprennent deux cas de maladie ganglionnaire diffuse à la première visite et un troisième patient qui présentait une seconde tumeur maligne non reliée.
Interventions : Études pour établir le stade d'évolution (examens hématologiques, radiographie, cartographie osseuse au technétium et tomographie par ordinateur), biopsies chirurgicales des lésions, chimiothérapie, radiothérapie et, dans certains cas, résection chirurgicale de la lésion.

From the Muskuloskeletal Oncology Unit, University of Toronto, Toronto, Ont.

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Reprint requests to: Dr. Robert S. Bell, Suite 476 E, Mount Sinai Hospital, 600 University Ave., Toronto, ON M5G 1X5

Principaux effets mesurés : Nombre de biopsies nécessaires au diagnostic et fréquence des complications nécessitant une intervention chirurgicale.
Résultats : Sept des 15 patients nécessitèrent plus d'une biopsie pour établir le diagnostic. Cinq patients eurent besoin de chirurgie pour des complications tardives comprenant des fractures pathologiques, des infections de plaie et de l'ostéonécrose. Après 24 mois, 13 patients étaient exempts de maladie et 2 étaient décédés.
Conclusions : Une biopsie et une évaluation pathologique appropriées sont essentielles au diagnostic du lymphome osseux. Ces mesures vont diminuer le besoin de répéter la biopsie. Il vaut mieux traiter le lymphome par des moyens médicaux. La chirurgie doit être réservée pour les biopsies et pour le traitement des complications.

Malignant lymphoma of bone is diagnosed in 2% to 5% of all patients with primary bone tumours and is associated with a better prognosis than other malignant lesions of bone.[1] The radiographic features of intraosseous lymphoma (a permeative pattern of bone lysis sometimes associated with an osteoblastic response) frequently mimic the findings associated with bone sarcoma,[2–6] and often the diagnosis of lymphoma is not considered before the initial biopsy. The differentiation of lymphoma from sarcoma generally requires special pathological techniques.[4] Appropriate handling of the initial specimen is critical to the pathological diagnosis. The management and prognosis of lymphoma are quite different from those of bone sarcoma, and the primary diagnostic work-up of patients with lymphoma is critical to their eventual management.

The treatment of patients with bone lymphoma by the orthopedic oncologist is frequently complicated by difficulty in making the initial diagnosis. This usually results from inappropriate biopsy before (and occasionally after) referral. We therefore reviewed all cases of bone lymphoma referred to the Muskuloskeletal Oncology Unit between 1984 and 1989. The details of diagnostic staging and biopsy were evaluated as well as the role of surgery in treating localized disease. This review demonstrates the frequency of failed initial biopsy in diagnosing primary lymphoma of bone and the excellent results obtained by nonsurgical management of this tumour. We conclude by recommending steps that may be taken to facilitate the diagnosis, staging and management of this uncommon malignant lesion of bone.

Patients and Methods

The hospital charts and office records of 18 patients with a diagnosis of malignant lymphoma of bone managed at our centre between 1984 and 1989 were reviewed. Specimens of all cases were reviewed by the same pathologist, whose report included the final diagnosis and the lymphoma subtype based on both the National Panel Working Formulation[7] and the Rappaport[8] classifications. Radiographs of all cases were reviewed. The diagnosis of primary osseous lymphoma was accepted when complete staging studies demonstrated no evidence of disease in extraskeletal sites or when bone symptoms preceded the discovery of occult extraosseous disease by at least 4 months.[2]

Of the initial 18 patients coded as suffering from intraosseous lymphoma, 3 were excluded: 2 patients with initial evidence of diffuse nodal disease on physical examination, and 1 patient who had a second unrelated malignant lesion, which complicated management and follow-up. For the remaining 15 patients we collected information related to the initial staging and biopsy (including the pre-biopsy diagnosis for eight patients referred to one of the authors, who listed a differential diagnosis prior to tissue sampling). Treatment and outcome were evaluated, and the effectiveness of surgery in treating the complications of nonsurgical therapy was assessed.

Findings

The mean age of the 15 patients (9 male, 6 female) was 42 years (range from 16 to 67 years) (Table I). Each patient was initially referred to our unit with the diagnosis of probable primary malignant disease of bone. A medical history was obtained and physical examination done, as well as routine staging studies appropriate to the work-up of a possible bone sarcoma (blood work, radiography of the lesion, technetium bone scanning, chest radiography and computed tomography of the lesion and the lungs). Magnetic resonance imaging was not routinely used in our centre before 1989 but is generally used today in the initial staging of this condition. None of the 15 patients had a history of lymphoid adenopathy, and no abnormal nodes were noted on physical examination. Two patients presented with B symptoms (weight loss, nights sweats, fever). All patients complained of localized pain, and in 10 patients there was a local mass or swelling. Routine hematologic and biochemical investigations gave normal results; however, six patients had an elevated erythrocyte sedimentation rate. Radiographs of

the local site showed lytic changes in seven patients, an osteoblastic response in one and mixed osteoblastic and lytic features in seven. A soft-tissue mass extending beyond the cortex was found on computed tomography in nine patients (Fig. 1). Technetium bone scanning demonstrated increased uptake at the local site in each instance, and in two patients questionable areas of increased isotope uptake at distant sites were evident at initial staging.

Eight of the patients were initially managed by one of the authors, who recorded the most likely diagnosis after examining the radiographic staging studies before biopsy. None of these eight patients was correctly characterized as having primary lymphoma. Two patients were thought to have metastatic carcinoma, two were incorrectly diagnosed as having probable chondrosarcoma, and individual tumours were diagnosed as most likely Ewing's sarcoma, giant cell tumour of bone, chronic osteomyelitis and osteosarcoma.

Six patients had undergone biopsy before referral to our centre. In each case, specimens were fixed in formalin and processed for routine histologic examination. Repeat biopsy of tissue at the tumour site was necessary in all patients to obtain material for more detailed pathological evaluation specific for lymphoma.

In one patient treated early in this series, the biopsy done before referral was interpreted as osteosarcoma. The lesion was resected, and lymphoma was identified in the resected specimen. In one case in which the initial biopsy was obtained at our centre, the sclerotic response to the tumour was such that adequate samples could not be obtained for pathological work-up of lymphoma. Gallium scanning identified a solitary abnormal mediastinal node, which provided adequate material for diagnosis on a second

Table I. Clinical Course of 15 Patients With Malignant Lymphoma of Bone

Age, yr	Sex	Site of disease	Histologic classification		Radiologic findings	No. of biopsies*	Preliminary treatment	Complications	Survival, mo
			WF	RAP					
42	M	Tibia	DLC	DH	L/B	3	C, R	—	87 DF
49	M	Sacrum	DLC	DH	L/B ST mass	1	C, R	—	54 DF
67	F	Clavicle	DLC	DH	L/B ST mass	2	C, R	—	51 DF
63	F	Femur	DSLC	DMHL	L	1	C, R	Fracture, open reduction, internal fixation, revision recurrence	29
26	M	Femur	DLC	DH	L	2	S	Fracture above allograft, allograft collapse	99 DF
54	M	Tibia	DLC	DH	L/B ST mass	2	C, R	—	72 DF
37	F	Fibula	DLC	DH	L	1	C, R	Wound infection	45 DF
16	M	Tibia	ImmB	DH	L/B ST mass	1	C, R	—	27 DF
61	M	Ischium	DLC	DH	L, pelvic ST mass	1	C	Acetabular fracture, total hip reconstruction	29 DF
54	F	Vertebral body (L3), A and P nodes, ovary	DLC	DH	L, fracture L3, 7 X 5-cm ovarian mass, hydronephrosis	2	C, R	L4 root symptoms	64 DF
29	M	Tibia, I and P nodes	DLC	DH	L/B	2	C, R	Pain—biopsy negative	25 DF
23	M	Tibia, bone marrow, spleen, thymus	DSLC	DMHL	L/B ST mass	1	C	Osteonecrosis of humerus	51 DF
25	F	Sacrum, M nodes	Hodgkin's nodular sclerosing		B ST mass	1	C	Pneumocystis carinii pneumonia	27 DF
59	M	Humerus, R, A and Ax nodes	DSC	DU	L	1	C, S, R	Failure of therapy	10
28	F	Femur, kidneys	DLC	DH	L ST mass	2	C	—	54 DF

*Number of biopsies required for diagnosis
A = abdominal, Ax = axillary, I = inguinal, M = mediastinal, P = pelvic, R = retroperitoneal, WF = International Panel Working Formulation, RAP = Rappaport classification, DLC = diffuse large cell, DSC = diffuse small cell, DSLC = diffuse small and large cell, DH = diffuse histiocytic, DU = diffuse undifferentiated, ImmB = immunoblastic, DMHL = diffuse mixed histiocytic and lymphocytic, L = lytic, B = blastic, ST = soft tissue, C = chemotherapy, R = radiotherapy, S = surgery, DF = disease free at the time of last follow-up

biopsy. In seven patients, therefore, the initial pathological studies failed to identify lymphoma.

According to the Rappaport classification, 11 patients had diffuse histiocytic lymphoma (diffuse large cell in 10, immunoblastic in 1 according to the Working Formulation classification), 2 patients had diffuse mixed histiocytic and lymphocytic tumours (diffuse small and large cell in the Working Formulation), 1 had diffuse undifferentiated lymphoma (small cell, noncleaved) and 1 patient had nodular sclerosing Hodgkin's disease. Two patients had high-grade lymphoma (those with diffuse undifferentiated and immunoblastic tumours) with the remainder being of intermediate grade. These subtypes are similar to those reported in other series.[2,6,9]

After the diagnosis of lymphoma had been made, all patients underwent further staging to evaluate the presence of extraosseous disease. Investigations included bone marrow biopsy, gallium scanning, computed tomography of the mediastinum, and computed tomography or lymphangiography of the abdomen. Occult extraskeletal involvement was recognized in six patients: three with nodal disease, two with lymphoma and one with visceral and nodal involvement. None of the patients had multiple osseous sites of disease.

A misdiagnosis of osteosarcoma was made in one patient who underwent resection and reconstruction at the primary tumour site; the other patients were treated without surgical resection. All eight patients with localized disease were given doxorubicin-based chemotherapy and local irradiation. Of the six patients with nonlocalized disease, all received chemotherapy, and three were treated with irradiation of the primary site. One underwent surgical stabilization at the time of biopsy.

There were two deaths. A 59-year-old man presented with a malignant lesion in his proximal humerus. Abdominal and retroperitoneal nodes were also involved. He had high-grade, diffuse, undifferentiated disease, which failed to respond to treatment, and he died 10 months from the time of diagnosis. The other, a 63-year-old woman, presented with an isolated lesion in the distal femur. She had intermediate-grade, diffuse, mixed histiocytic and lymphocytic lymphoma. She sustained a pathologic fracture during her course of radiotherapy 6 months after her initial diagnosis and subsequently died of recurrent disease 23 months later. The remaining 13 patients have been followed up for an average of 49 months (range from 25 to 99 months) and have no clinical or radiographic evidence of recurrent disease.

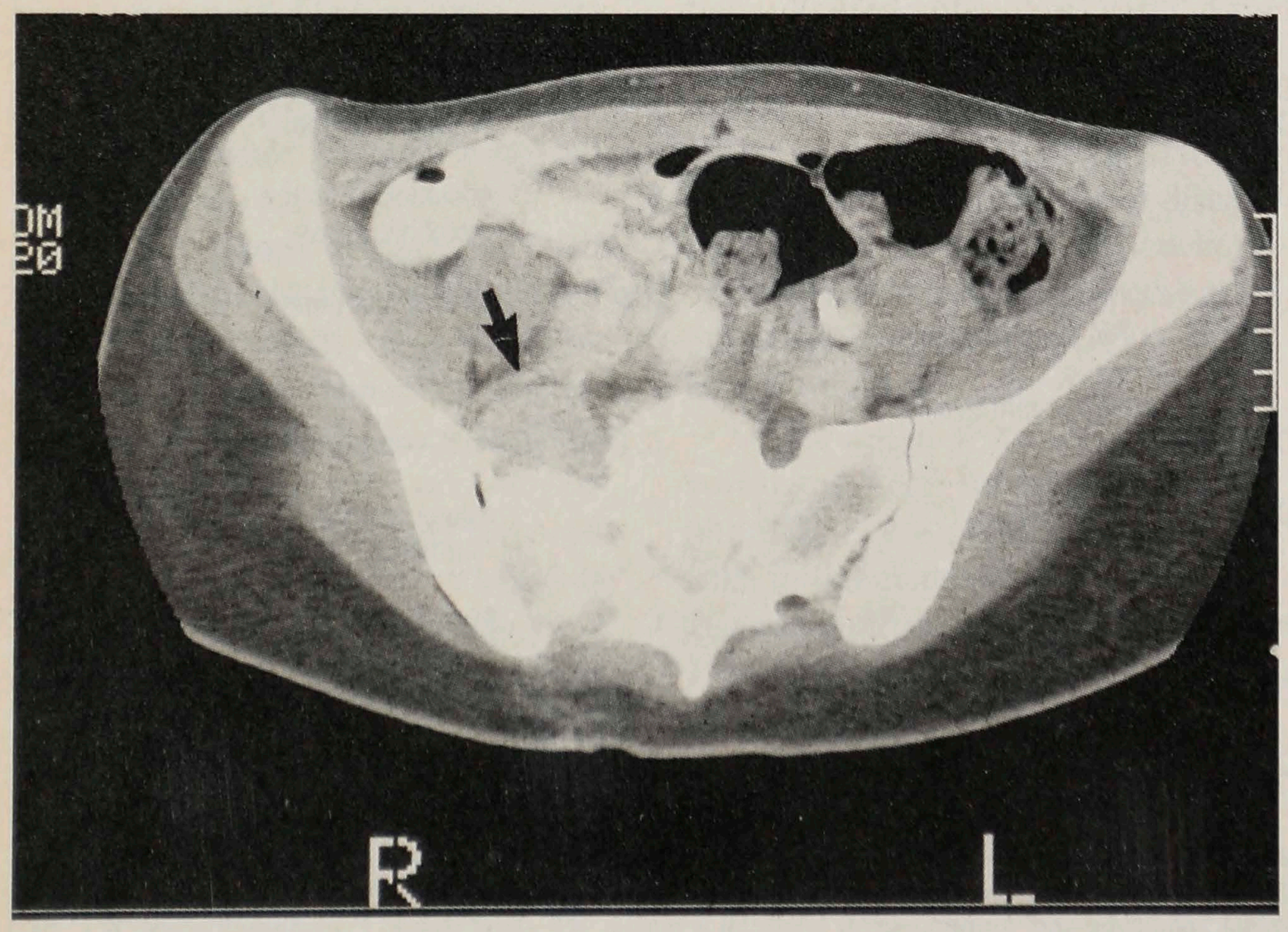

FIG. 1. Computed tomography scan demonstrating large soft-tissue mass (arrow) extending from right sacrum that is in difficult location for biopsy.

Secondary surgical procedures were required in five patients. The patient who underwent surgical resection and allograft bone reconstruction of a femoral lymphoma (initially diagnosed as osteosarcoma) sustained two allograft fractures requiring reoperation. His functional outcome at 8-year follow-up is poor. The 63-year-old woman who presented with a highly lytic distal femoral lesion and sustained a pathologic fracture during radiotherapy treatment of the local site required internal fixation of the fracture. The fracture had not united 4 months after fixation, and the femur required replacement with a prosthesis 8 months after initial diagnosis. Intraoperative biopsies at the local site showed only necrotic tumour; however, she subsequently died of recurrent disease. One patient suffered a pathologic fracture of the right acetabulum 4 months after the completion of chemotherapy for pelvic lymphoma (Fig. 2). At the time of acetabular reconstruction, extensive biopsies showed no evidence of recurrent disease, and the patient was disease free at a 29-month follow-up (Fig. 3).

Two patients complained of continued local pain more than 6 months after completion of medical

and radiation treatment. One patient with a fibular lesion complained of pain at the biopsy site. On exploration a deep wound infection was found, which resolved with drainage and antibiotics. The patient was disease free 45 months after treatment. One patient with a humeral lesion complained of persistent shoulder pain associated with a "hot" bone scan 12 months after treatment. Biopsy showed osteonecrosis of the humeral head. The humerus was not reconstructed, and the patient remained disease free with good shoulder function after 51 months.

All patients required careful surveillance in the post-treatment phase. Radiographs and bone scans of the local site remained abnormal (Fig. 4), so any increase in pain at the local site was investigated with open or needle biopsy. Recently, regression of the marrow disease at the local site has been monitored by magnetic resonance imaging. The patient who died of recurrent disease after responding to initial treatment was found to have increased signal intensity in the marrow on magnetic resonance imaging, sug-

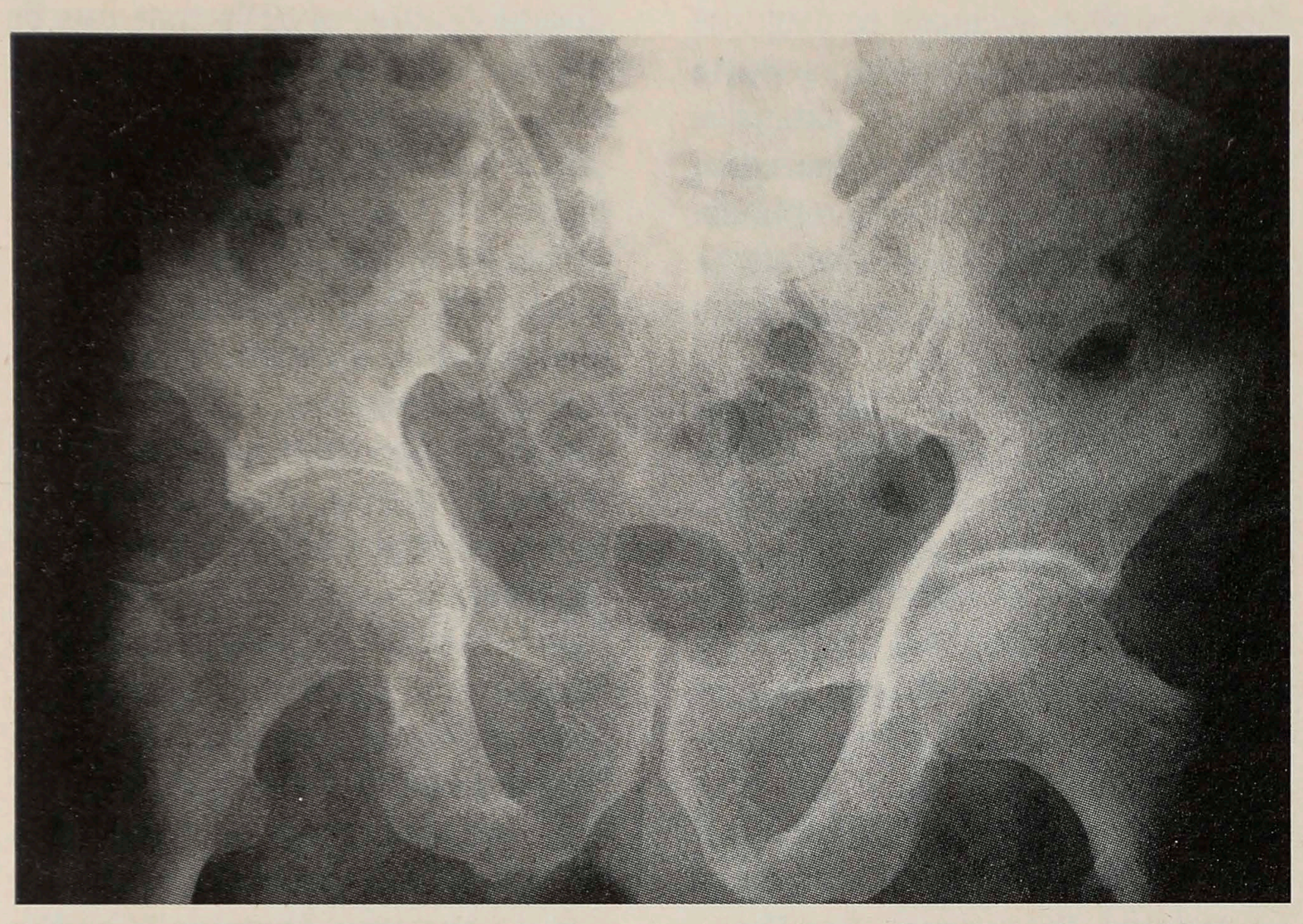

FIG. 2. Radiograph shows pathologic fracture of right acetabulum after therapy for bone lymphoma.

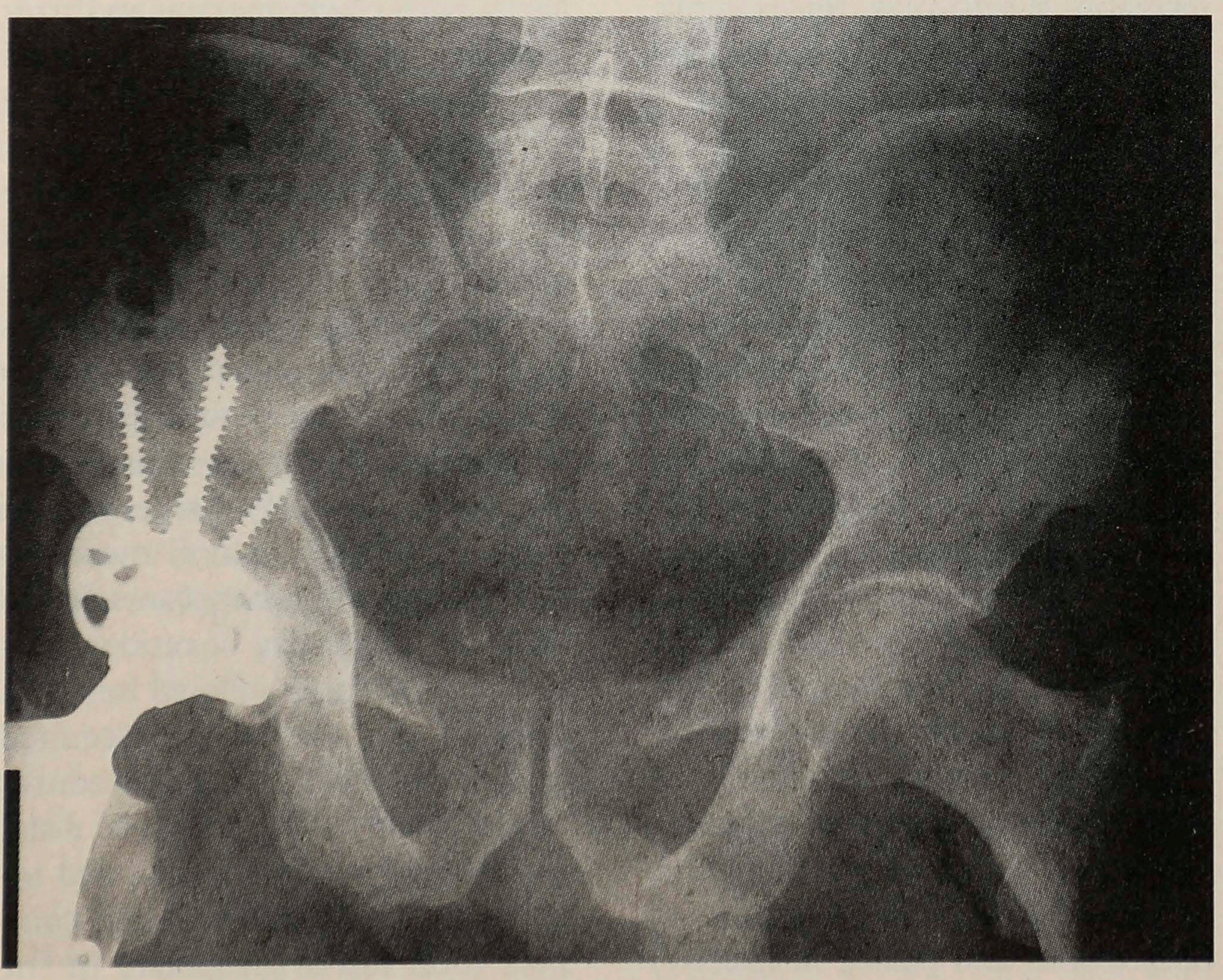

FIG. 3. Reconstruction of hip revealed no evidence of tumour.

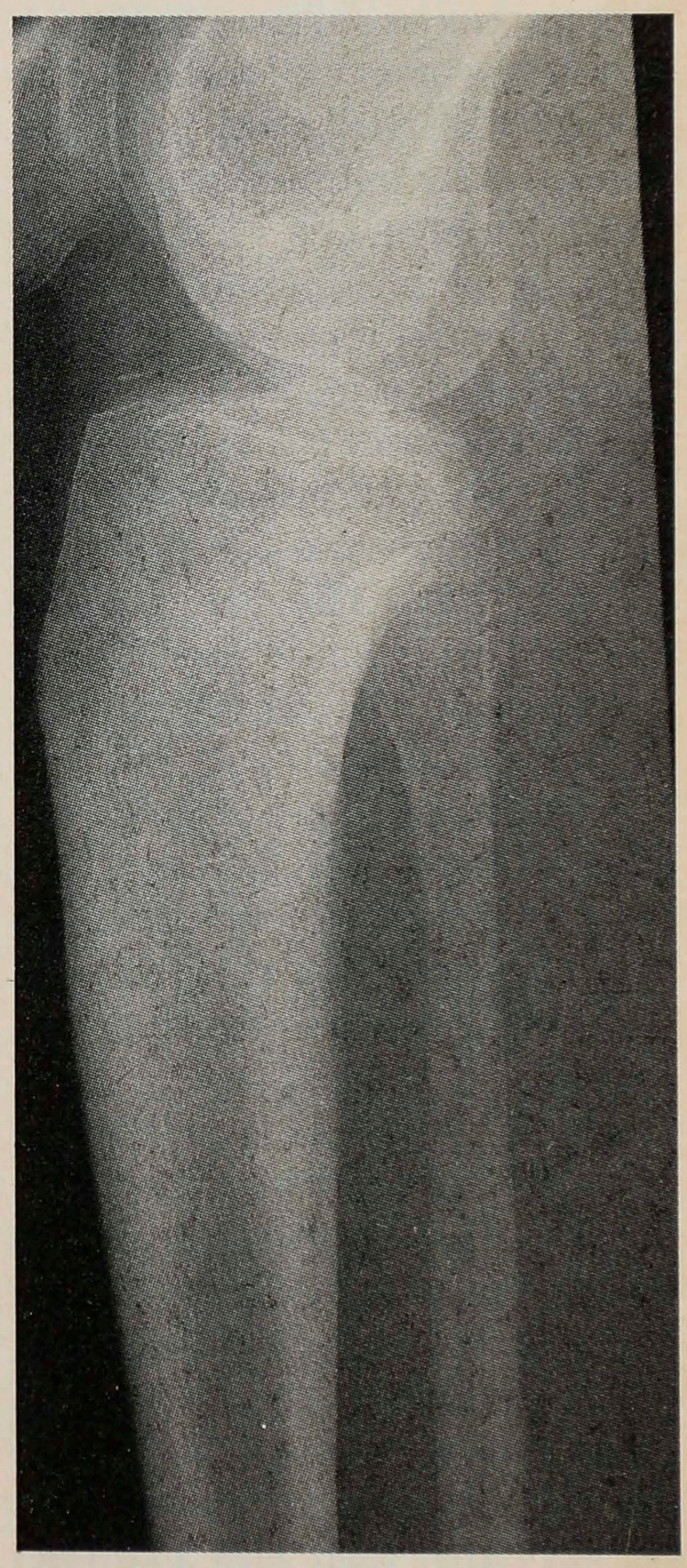

FIG. 4. Two years after treatment for osseous lymphoma of tibia. Persistent radiographic changes of mixed lysis and sclerosis with pain necessitated repeat biopsy, which was negative for recurrent disease.

gestive of recurrent disease. In addition to surveillance of the local site, patients were followed up with nodal examination, gallium scanning and computed tomography of the chest and abdomen when applicable.

Discussion

Although lymphoma of bone is relatively rare, its recognition is critical since it mimics bone sarcoma in its clinical features yet is managed entirely differently. Unlike sarcoma, surgical resection of the primary tumour is not necessary, and most patients achieve complete remission with medical or radiation treatment. The most important finding in this review of 15 patients with bone lymphoma relates to the frequency with which lymphoma is overlooked at the time of initial assessment and the frequency with which the initial biopsy fails to establish a diagnosis. In all eight cases treated primarily by an experienced orthopedic oncologist, lymphoma was not considered the most likely diagnosis prior to biopsy. This is important, because failure to include lymphoma in the differential diagnosis may result in mishandling of the specimen obtained at biopsy. This was the case in 7 of the 15 patients reviewed in this series, all of whom required secondary biopsies to establish the diagnosis.

The diagnosis of lymphoma is best served by obtaining tissue without the crushing artefact that alters the cellular morphology. In obtaining material from a bone lesion, it is best to sample the extraosseous soft-tissue mass if present and to obtain the specimen with sharp scalpel dissection rather than with a curette. Avoidance of a cortical window is also recommended if a soft-tissue mass is present to avoid creating a stress concentration area in the bone, which may fracture with later radiotherapy. If absence of a soft-tissue mass necessitates opening the cortex to obtain intraosseous tumour, gentle removal of the tumour with a periosteal elevator is preferable to scraping out the specimen with a curette. Although needle biopsy is often adequate to diagnose primary bone tumours presenting with a soft-tissue mass or extensive bone lysis, the studies required to confirm the diagnosis of lymphoma necessitate incisional biopsy of the tumour to provide the quantity necessary.

Frozen section examination of all bone biopsies should be done before wound closure to ensure that adequate material has been obtained. If the frozen section shows a round cell tumour, it is encumbent on the surgeon to ensure that the handling of the specimen will permit the diagnosis of lymphoma. In addition to ensuring that the frozen section demonstrates non-necrotic diagnostic material, the surgeon should encourage the pathologist to utilize other fixative agents in addition to formalin. B-5 fixative, for example, preserves cytologic detail better than formalin for lymphoma. In differentiating round cell malignant lesions of bone, the pathologist is faced with a differential diagnosis that includes metastatic disease (small cell lung carcinoma, melanoma) and primary bone malignancies (Ewing's sarcoma, myeloma, small cell osteosarcoma, peripheral neuroectodermal tumour) as well as lymphoma. Immunohistochemical markers specific for mesenchymal cells (vimentin), epithelial cells (keratin), leukocytes (common leukocyte antigen), melanin, neural differentiation (neuron-specific enolase) are often critical in differentiating these lesions. It is therefore necessary that specimens of viable nonossified tumour are both frozen and placed in fixatives appropriate for immunohistochemistry (for example Omnifix). Electron microscopy is frequently useful in differentiating round cell tumours, and a specimen should be preserved in glutaraldehyde for this purpose. Finally, fresh tissue should be placed in tissue culture medium to permit disaggregation of cells that can be processed for flow cytometric analysis of fluorescent antibodies to lymphocyte antigens. Ensuring that sufficient tissue is available and processed for all of these studies increases the likelihood that the initial biopsy will be sufficient for the diagnosis of lymphoma.

Once the diagnosis has been made, the patient should be referred to an oncologist for consideration of medical or radiation therapy, or both. There is controversy in the literature as to whether localized bone lymphoma can be treated with radiation alone and whether patients with extraosseous disease should undergo radiotherapy at the local site in addition to systemic therapy.[1,4,5,9–12] We generally advise the patient with isolated or more diffuse disease to undergo both chemotherapy and irradiation of the local site. As documented in this review, combined management results in a complete and prolonged disease response in most patients with intermediate-grade disease.

The surgical management of intraosseous lymphoma is restricted to the treatment of complications of nonsurgical therapy. In this review the indications for surgery were pathologic fracture and investigation of persistent symptoms at the local site suggestive of residual disease. Two patients suffered fractures and both of these fractures required complex reconstructive procedures (replacement of the destroyed distal femur and knee in one case and acetabular reconstruction with complex total hip arthroplasty in the other). The reasons for pathologic

fracture are easily recognized. In virtually all cases lymphoma presents with evidence of extensive bone lysis. Frequently a cortical window is necessary to obtain tumour tissue. This cortical defect acts as a stress concentration focus, and it has been demonstrated that these defects do not remodel normally if treated with radiotherapy.[13] Despite the risk of pathologic fracture, we do not recommend prophylactic fixation of the primary site at the time of initial biopsy. As noted in this series, it is rare that a definitive diagnosis of lymphoma is made on frozen section examination of the initial biopsy. The extensive dissection necessary for fixation of a lytic lesion is contraindicated in cases of bone sarcoma (Ewing's sarcoma or small cell osteosarcoma), since extension of the disease into the surrounding tissues may obviate the potential for limb salvage surgery. We therefore advise that patients with lytic tumours at risk for fracture (especially in the lower extremity) be protected from mechanical loading until after medical and radiation treatments have been completed. It is important that treatment by a physical therapist is initiated during the period of medical treatment to ensure that limb function is maintained and maximized. Some lytic lesions may show progressive ossification during treatment, eliminating the risk of fracture. If bone healing does not occur, stabilization of the lesion can be accomplished after nonsurgical treatment is completed. Discussion with the radiation oncologist regarding the preferred surgical approach for fixation may avoid inclusion of the operative site in the treatment volume.

The second indication for surgery relates to the possibility of persistent or progressive disease at the local site despite treatment. The surveillance of disease at the initial site is difficult since plain radiographic, bone scan and magnetic resonance abnormalities generally persist despite elimination of the tumour. Careful analysis of symptoms is therefore necessary. Patients with persistent symptoms have residual disease, infection, mechanical instability and, rarely, osteonecrosis. All of these possibilities require exploration for diagnosis and possibly treatment. We therefore recommend repeat biopsy in patients with persistent or recurrent symptoms.

Conclusions

Although lymphoma of bone is found in fewer than 5% of patients with primary bone tumours, it is an important diagnosis to consider in patients with lytic or mixed lytic and blastic primary bone lesions. The diagnosis of primary bone lymphoma requires that material is obtained for a variety of specific pathological tests, and initial processing of material is critical to this process. Medical and radiation treatment of this disease is generally successful, and surgical management should be reserved for biopsy, the treatment of complications and the diagnosis of recurrent disease.

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Craniocervical Fusion With Contoured Luque Rod and Autogeneic Bone Graft

Peter M. Ellis, MD, CM, MSc, FRCSC;* J. Max Findlay, MD, PhD, FRCSC†

Objective: To examine the efficacy of internal craniocervical fixation with a Luque rod and autogeneic bone graft for craniocervical instability.
Design: A case series.
Setting: A university-affiliated hospital.
Patients: Six patients with craniocervical instability from diverse causes.
Interventions: Craniocervical fusion with a custom-formed Luque rod wired to the occiput and a variable number of vertebrae overlaid with autogeneic bone graft, followed by bracing with either a halo vest or a removable, stiff, plastic cervical orthosis.
Main Outcome Measures: Caniocervical fusion and neurologic stability.
Results: All patients maintained good craniocervical alignment. Radiologic bony fusion was achieved in five patients. Three patients remained neurologically stable and three had improved neurologic status.
Conclusion: Craniocervical fusion with the contoured Luque rod and autogeneic bone grafting, usually in combination with a temporary plastic cervical orthosis, is of value in managing craniocervical instability.

Objectif : Examiner l'efficacité de l'arthrodèse craniocervicale interne à l'aide d'une tige de Luque et d'une greffe osseuse autogène dans les cas d'instabilité craniocervicale.
Conception : Série de cas.
Contexte : Hôpital affilié à une université.
Patients : Six patients atteints d'instabilité craniocervicale attribuable à des causes diverses.
Interventions : Arthrodèse craniocervicale effectuée à l'aide d'une tige de Luque sur mesure fixée à l'occiput par des fils et du recouvrement d'un nombre variable de vertèbres d'une greffe osseuse autogène, suivie d'une immobilisation à l'aide d'un corset de fixation à couronne (halo) ou d'une orthèse cervicale en plastique rigide amovible.
Principaux effets mesurés : L'arthrodèse craniocervicale et la stabilité neurologique.
Résultats : Tous les patients ont conservé un bon alignement craniocervical. Des radiographies ont démontré une fusion osseuse chez cinq patients. L'état neurologique de trois patients est demeuré stable et celui de trois autres s'est amélioré.
Conclusion : L'arthrodèse craniocervicale effectuée à l'aide d'une tige de Luque profilée et d'une greffe osseuse autogène, habituellement jumelée au port d'une orthèse cervicale temporaire en plastique, aide à traiter l'instabilité craniocervicale.

Although less common than at other spinal levels, instability at the craniocervical junction can occur from many causes, including trauma, degeneration, surgery and neoplasia. The potential neurologic sequelae may be catastrophic, and internal surgical stabilization is often required.

The problems of occipital–cervical instability and swan-neck deformity requiring craniocervical stabilization have resulted in a number of fusion techniques, with or without internal fixators or rigid external immobilization, and with or without bone grafting.[1–11] Older methods that utilized small-gauge wire or provided inadequate postoperative immobilization resulted in failure of fusion in over 20% of cases.[11] With these techniques, immobilization for less than 6 to 12 months has been associated with a greater chance of nonunion, malunion or increased neurologic deficit. In this report, we describe six pa-

*From the *Division of Neurosurgery, Queen's University, Kingston, Ont., and the †Division of Neurosurgery, The University of Alberta, Edmonton, Alta.*

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Reprint requests to: Dr. J. Max Findlay, 2D1.02 Walter C. Mackenzie Health Sciences Centre, 8440–112th St., Edmonton, AB T6G 2B7

tients with craniocervical instability from various causes, who were all successfully treated by a new method of internal craniocervical fixation with a Luque rod overlaid with autogeneic bone graft.

Patients

The patients ranged in age from 2 to 72 years (mean 34.3 years), and the follow-up ranged from 9 to 53 months (mean 28.3 months). The clinical courses of the six patients are shown in Table I.

Surgical Technique

If craniocervical instability is acute or highly unstable, cervical traction is maintained with Gardner–Wells tongs until the patient is brought to the operating room, where fibreoptic intubation is performed with the patient awake. The patient is placed prone on the operating table, the head is secured with pin fixation under local anesthesia, and a cervical x-ray film is obtained and a neurologic examination done to ensure correct craniocervical alignment before the induction of general anesthesia. We monitor patients intraoperatively with somatosensory evoked potentials.

A midline, posterior cervical and low occipital incision is made from the inion to the spinous process of C7, and soft tissues are reflected off the occiput below the superior nuchal line as well as all laminae from C1 to C4 or C5. A burr hole is made on either side of midline in the suboccipital region. Smaller holes are drilled adjacent to these, and flexible, monofilament stainless-steel wires (1.0 mm diameter; Synthes Co., Waldenburg, Switzerland) are looped between them, beneath the skull. Sublaminar wires are passed bilaterally at C1 and all distal levels to be included in the fusion. If a laminectomy has been performed, the wires are passed through holes drilled through the bony edge of the laminectomy, which is usually the medial aspect of the facet joint. The suboccipital area, laminae and spinous processes are decorticated with a high-speed air drill. The contoured Luque rod (3/16 inch diameter; Zimmer S.A., Vitry Cedex, France) is then inserted and secured to occiput and spine with the wires, which are either looped around the rod or passed through small holes in the rod (Fig. 1). The craniocervical junction is fixed in a neutral position. The angle between the cranial loop of the construct and the paired vertical bars varies and is best determined and formed preoperatively on the basis of postreduction x-ray films. The angle can be modified intraoperatively with a Luque rod bender.

Morcellated cancellous and cortical bone from the iliac crest is then placed over decorticated bone surfaces, and the wound is closed. Postoperatively, the patient is kept in either a rigid, plastic cervical orthosis or, under certain circumstances, a halo vest (Bremer Medical Co., Jacksonville, Fla.) for 3 to 4 months. We

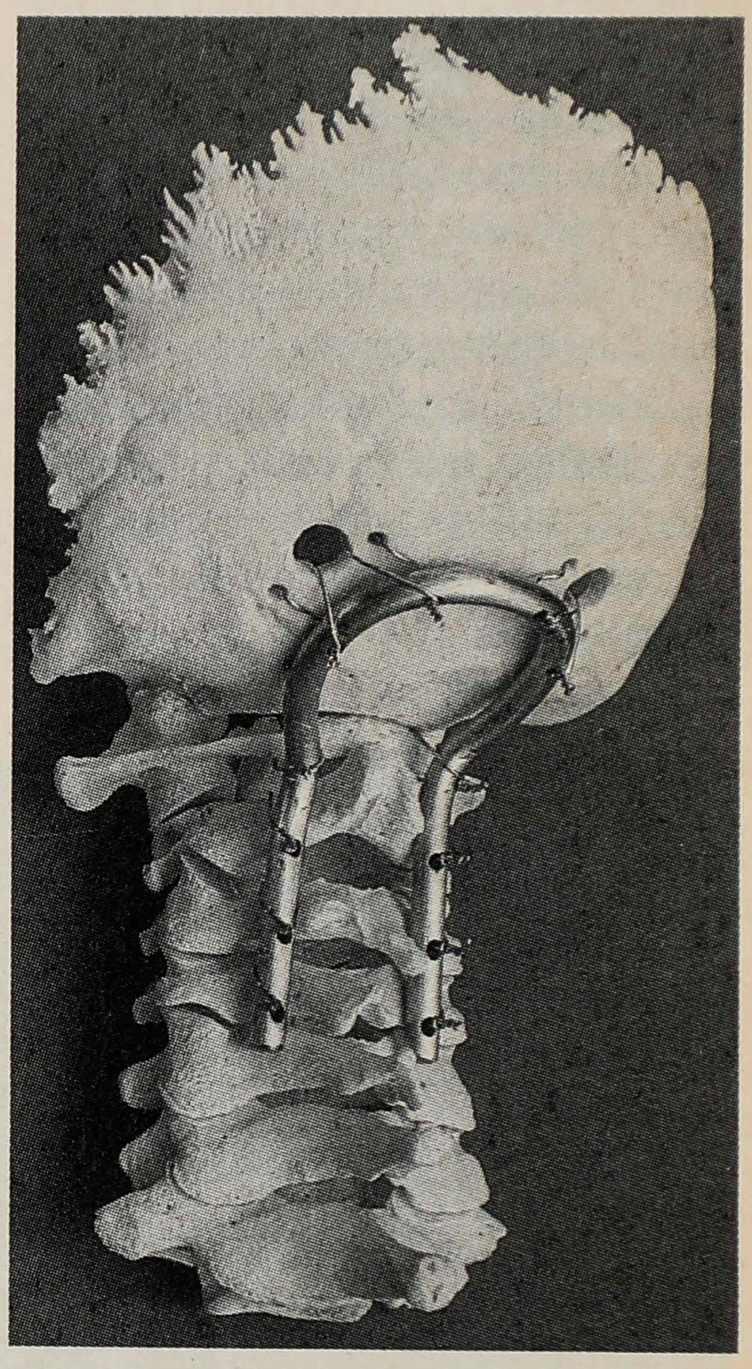

FIG. 1. Model of contoured Luque rod in occiput-to-C4 fusion.

Table I. Clinical Course of Six Patients Who Underwent Craniocervical Fusion With the Luque Rod and Autogeneic Bone Grafting

Patient no.	Age, yr	Sex	Cause of craniocervical instability	Preoperative status	Levels fused	Neurologic outcome	Complications	Follow-up, mo
1	40	F	Trauma, MVA	Quadriparesis	Oc–C4	Improved, ambulatory	None	53
2	15	M	Postlaminectomy	Swan-neck, dysphagia	Oc–C6	Improved, normal	None	34
3	48	F	Rheumatoid disease	Quadriparesis	Oc–C5	Improved, non-ambulatory	None	31
4	77	M	Prostate metastasis	Fixed flexion deformity	Oc–C4	Unchanged, normal	None	29
5	2	F	Trauma, MVA	Quadriplegia	Oc-C2	Unchanged, tetraplegia	Superficial wound infection	14
6	72	M	Trauma, MVA	Normal	Oc–C2	Unchanged, normal	None	9

F = female, M = male, MVA = motor-vehicle accident, Oc = occiput, C = cervical

fused fewer vertebral levels later in our series.

Results

Patient 1 was rendered severely quadriparetic as a result of a motor-vehicle accident, which caused atlanto-occipital dislocation. She underwent craniocervical fusion (occiput to C4) after a week of light (2 kg) cervical traction and was immediately mobilized in a Philadelphia collar, which she wore for 4 months. She made a good neurologic recovery, becoming ambulatory with only mild dysphonia, dysphagia and limb spasticity.

Patient 2 presented with basilar invagination and a severe swan-neck deformity (flexion of the lower cervical spine and hyperextension at the craniocervical junction),[12] after laminectomy and syringosubarachnoid shunting for a Chiari malformation and associated syringomyelia. After 1 week of cervical traction (8 kg) and significant reduction of his deformities, he underwent an occiput-to-C6 fusion followed by 3 months in a halo vest.

Patient 3, who suffered from long-standing, severe rheumatoid arthritis and worsening quadriparesis, was found to have a swan-neck deformity, atlanto-occipital, atlantoaxial and subaxial subluxations and rheumatoid pannus compressing the upper spinal cord. She underwent laminectomy of C1 and C2 followed by an occiput-to-C4 fusion.

Patient 4, who had metastatic prostate carcinoma, presented with acute neck pain from a pathologic C2 (odontoid) fracture-subluxation (Fig. 2). Occiput-to-C4 fusion was performed after traction reduction. The patient's condition was stable and he remained without neurologic deficit at a follow-up of 2.5 years.

Patient 5 was a 2-year-old child who suffered atlanto-occipital dislocation and complete quadriplegia in a motor-vehicle accident (Fig. 3). Occiput-to-C2 fusion with morcellated occipital calvarial bone (Fig. 4) was complicated by a temporary and superficial wound infection but allowed early wheelchair mobilization (Fig. 5).

Patient 6 was a 72-year-old man who suffered a combination of a C1 burst (Jefferson's) fracture and an odontoid-base fracture-subluxation in a motor-vehicle accident. After traction reduction an occiput-to-C2 fusion was performed, and the patient remained without any neurologic deficit.

Only one patient in this series (patient 2) was placed in a halo vest

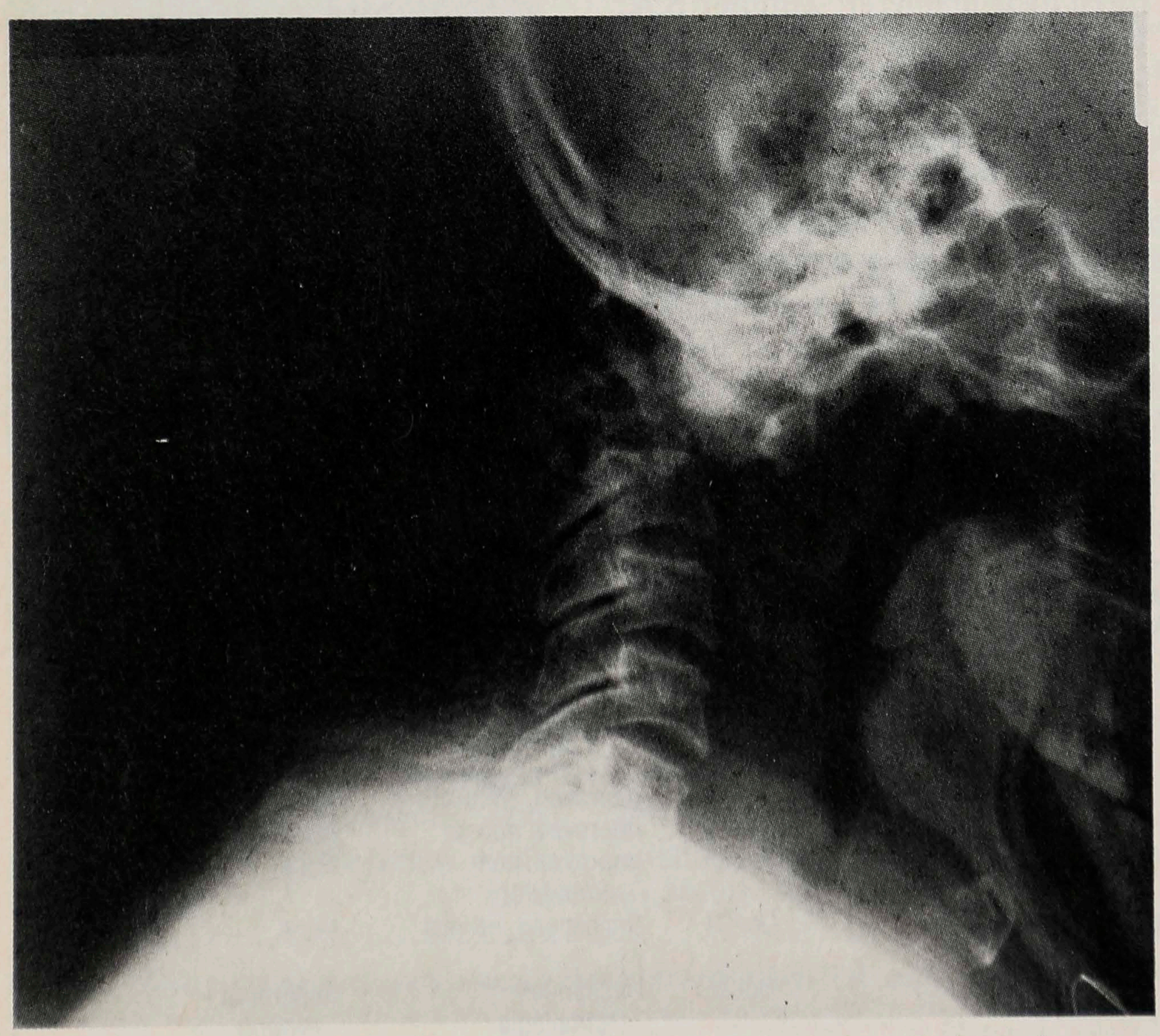

FIG. 2. Lateral cervical x-ray film of 77-year-old man with pathologic fracture-subluxation of C2, secondary to prostate carcinoma metastasis (patient 4). After traction reduction, patient underwent occiput-to-C4 Luque rod fusion and remained neurologically intact.

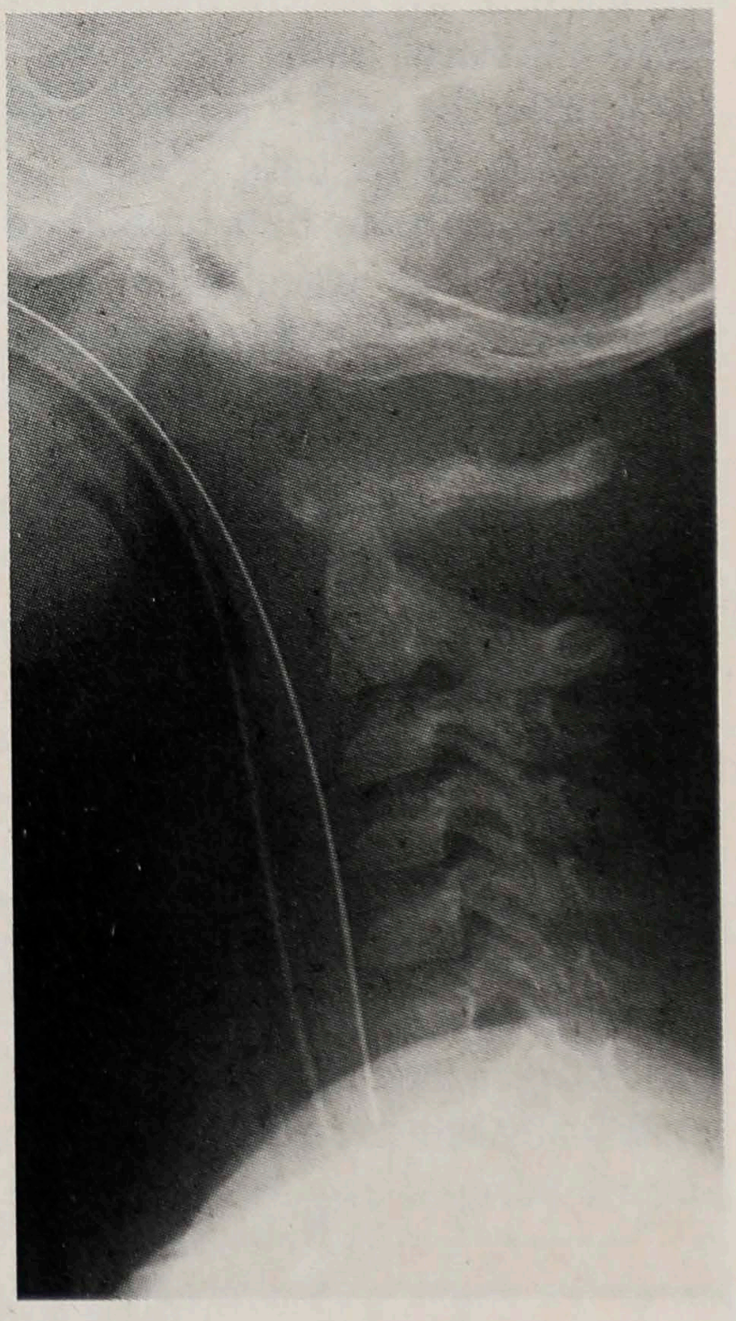

FIG. 3. Lateral cervical x-ray film of 2-year-old child who suffered traumatic atlanto-occipital dislocation (patient 5). Note widened space between occiput and atlas.

postoperatively. The other five were treated in a stiff plastic cervical collar, such as a Philadelphia collar, for 3 months after craniocervical fusion. Restored craniocervical alignment was maintained in all patients. Alignment and fusion were documented radiographically in every patient except the one with rheumatoid disease, in whom alignment was maintained but bony fusion was documented. No malunion or nonfusion was seen.

Discussion

Ransford and colleagues[13] used contoured metal loop fixation with autogeneic iliac bone graft for various lesions in patients with gross craniocervical instability after posterior and anterior decompression at the foramen magnum. We used a similar technique to correct and stabilize the craniocervical junction in cases of traumatic atlanto-occipital and atlantoaxial dislocation, rheumatoid instability, pathologic fracture due to metastasis and progressive swan-neck deformity and basilar invagination after laminectomy for syringomyelia.

FIG. 4. Patient 5. Intraoperative photograph showing internal fixation of occiput to C2 with contoured Luque rod surrounded and overlaid with calvarial cancellous and cortical bone graft harvested from patient's occiput exposed during operation.

The atlanto-occipital junction forms a functional complex with the atlantoaxial joint. It consists of a double condylar joint allowing flexion and extension but little lateral bending or axial rotation. The occipital condyles are convex and articulate with the cup-shaped superior articular surfaces of the atlas. Although this joint deepens with age, the true stability of the craniovertebral junction is derived from the ligaments and muscles.[14] The ligamentous supports are in two functional groups: the atlanto-occipital ligaments (anterior and posterior) and the axial-occipital ligaments, consisting of the apical and alar ligaments as well as the tectorial membrane.[15–17] Traumatic,[14,15,18–22] surgical,[1,2,12,17,23–25] inflammatory[3,26–30] or neoplastic[4,31–34] disruption of these bony structures or their ligamentous supports, resulting in instability of the craniovertebral junction, requires operative stabilization.

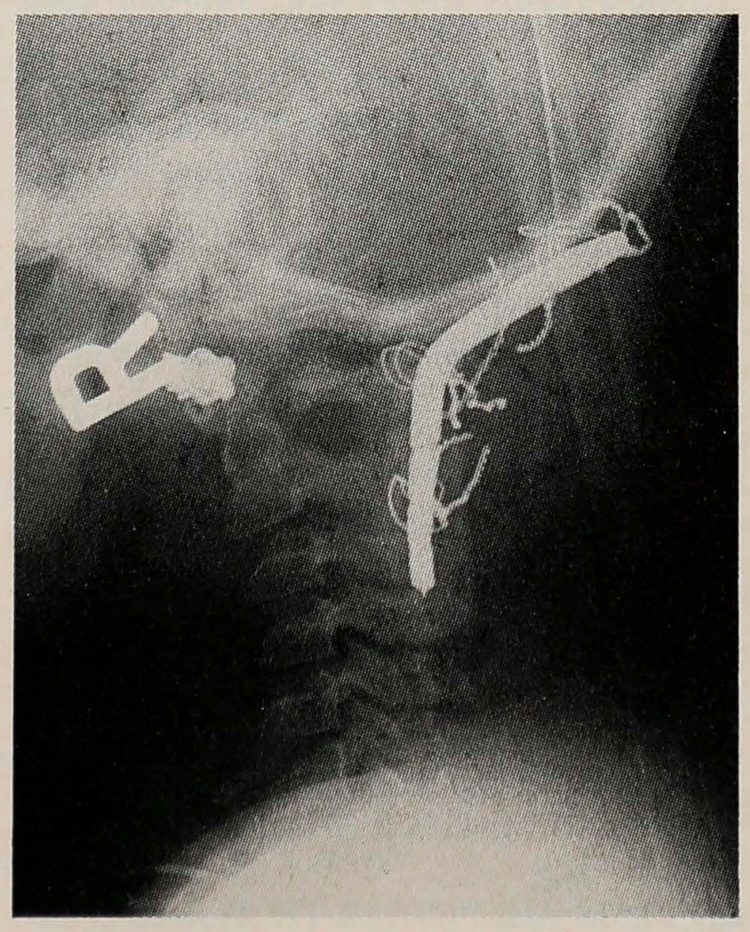

FIG. 5. Patient 5. Lateral cervical spine x-ray film 4.6 months postoperatively, showing occiput-to-C2 fusion with combination of Luque rod and autogeneic bone.

Several radiologic criteria have been proposed to diagnose atlanto-occipital dislocation.[3,15,35] The most common radiologic finding is a widened retropharyngeal soft-tissue thickness anterior to the C1–2 level.[5] However, the most consistent criterion in the common anterior dislocation is a ratio greater than 1.0 of basion, posterior arch C1:opisthion, anterior arch C1, as determined from lateral radiographs, magnetic resonance images or computed tomography scans. The ratio is 0.8 in normal alignment.[36]

Treatment for atlanto-occipital dislocation includes airway management, realignment and immobilization, then fusion when the patient's condition is stable.[36] Laminectomy may be indicated in the presence of a progressive neurologic deficit and a documented compressive lesion that can be removed or relieved by posterior decompression. Atlanto-occipital dislocation is a pure ligamentous injury, and persistent instability after prolonged immobilization can be expected; subluxation on flexion after 5 months in a four-poster brace has been reported.[37]

Instability at the upper cervical and craniocervical levels has most commonly been managed surgically, with a combination of sublaminar wires and iliac bone strips or formed cortical tibial or rib struts.[1,2,7,12,23] The latter technique involves the harvest of autogeneic cortical bone that is shaped to approximate anatomical configuration with the posterior cervical lordosis and then wired in one piece to the laminae.[2]

Occipitocervical fusion with the Luque rod has been reported for rheumatoid disease with basilar impression and craniocervical instability.[28] Bone fusion was seen radiographically at about 4 months in 12 of 13 patients. Halo immobilization was not used later in this series owing

to the extremely rigid stabilization afforded by the internal fixator alone.

Chordoma of the axis, basilar impression from osteogenesis imperfecta and occipitalization of the atlas constituted the lesions in Ransford's series.[13] All of these patients required both anterior and posterior craniocervical decompression, and the Luque rod and bone fusion provided the necessary postoperative stabilization. The patients were managed in a cervical collar or cervical brace with excellent results. The authors recommended the use of cancellous bone chips, internal metal fixation and sublaminar wires. Autogeneic bone graft may have a faster fusion rate with a lower chance of nonunion,[38] although there are no series comparing allograft with autogeneic graft for this sort of fusion.

The method reported here is relatively simple, providing immediate and rigid stabilization with internal Luque rod fixation. Five of the six patients required no halo-vest stabilization postoperatively. This allows a morcellated cortical and cancellous graft to be used to ensure good bony union, as documented radiographically, and is possibly a superior method of fusion for severe instability at the craniocervical junction. In two recent papers[39,40] presenting a similar technique of fusion the authors have described good success in similar patient groups.

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Disseminated Intravascular Coagulation Complicating Gastric Perforation in a Bulimic Woman

Glen S. Roseborough, MD;* William A. Felix, MD, FRCSC†

The authors report the case of a 43-year-old woman who presented with an acute abdomen and in shock after a bulimic episode. The fatal outcome in this case was due to disseminated intravascular coagulation (DIC). The death represents the fifth reported in the literature among bulimic or anorexic patients who sustained a gastric perforation. This is the first documented case of DIC in a bulimic patient who died. The other reported cases are reviewed, and the findings are compared with those in this case.

Les auteurs signalent le cas d'une femme de 43 ans qui fut reçue en abdomen aigu et en état de choc après une crise de boulimie. Dans ce cas, le décès fut causé par une coagulation intravasculaire disséminée (CID). C'est le cinquième décès relié à une perforation gastrique rapporté dans la presse médicale chez des malades boulimiques ou anorexiques. C'est le premier cas démontré de CID chez une patiente boulimique qui est décédée. Les autres cas signalés sont passés en revue et comparés à celui-ci.

Acute gastric dilatation and perforation is a rare complication of bulimia but one that can have disastrous consequences. We report the case of a bulimic woman who died of disseminated intravascular coagulation, which developed as a result of gastric perforation after an acute bulimic episode.

Case Report

A 43-year-old woman with no medical history was brought to the emergency department after a relative found her at home in acute distress. She had been perfectly well that morning but became hypotensive en route to the hospital and presented in shock with an acute abdomen.

The patient was thin and was acutely distressed, with a massively distended and rigid abdomen. She was tachycardic and her systolic blood pressure ranged from unattainable to 60 mm Hg. The lower limbs were mottled and blue, although pulses were palpated in all limbs. Repeated attempts to pass a Foley catheter failed due to an apparent obstruction. Initial blood work showed a leukocyte count of 6.74×10^9/L, a hemoglobin level of 139 g/L and a platelet count of 298×10^9/L. Serum electrolyte levels were as follows: sodium 153 mmol/L, potassium 3.2 mmol/L, chloride 102 mmol/L, total carbon dioxide 27 mmol/L, with an anion gap of 26 mmol/L. Arterial blood gas analysis revealed a pH of 7.27, a partial pressure of oxygen of 292 mm Hg (on 100% oxygen), a partial pressure of carbon dioxide of 48.3 mm Hg, and a bicarbonate level of 22.0 mmol/L. The prothrombin and partial thromboplastin times were 10 and 22 seconds respectively.

The patient was resuscitated with intravenous fluids. Laparotomy revealed a massively dilated stomach, extending from the xiphoid to the pubis, that filled the entire abdominal

From the Department of Surgery, Memorial University of Newfoundland, St. John's, Nfld.

**Former rotating intern, Memorial University of Newfoundland, St. John's*

†Chief of surgery, St. Clare's Mercy Hospital, St. John's

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Reprint requests to: Dr. Glen S. Roseborough, 2830 Murray Dr., Victoria, BC V9A 2S5

cavity and ballooned out of the wound. After the stomach was decompressed the patient was found to have several litres of food in the abdominal cavity that spilled out of the stomach through a 2-cm long laceration on the lesser curvature just below the gastroesophageal junction. The stomach was resected, a Roux-en-Y anastomosis was performed and the abdominal cavity was irrigated with several litres of warm saline.

Intraoperatively the duodenum was also noted to be grossly dilated to the ligament of Treitz, and the entire small bowel was white with lymphatic congestion. There was no evident cause of obstruction, and the rectum was empty. During the course of the procedure, hemostasis was difficult to achieve, and it was suspected intraoperatively that disseminated intravascular coagulation (DIC) was developing. Eventually, however, hemostasis was achieved to the point at which the abdomen could be closed with suction drains in place.

Postoperatively in the intensive care unit the patient remained extremely unstable and continued to need massive volume support. During resuscitation the platelet count dropped to 8×10^9/L, with a prothrombin time of 18 seconds, a partial thromboplastin time of more than 400 seconds and a fibrin-degradation-products level of more than 40 µg/L, confirming the diagnosis of DIC. In addition to 6 units of packed cells received in the operating room, the patient was transfused with 15 units of packed cells, 40 units of platelets and 10 units of fresh frozen plasma in the intensive care unit. Despite this treatment, intravenous antibiotics and a final trial of heparin therapy, acute respiratory distress syndrome developed, and the patient began to bleed through the wound, nose, rectum and endotracheal tube in quantities exceeding replacement volumes. The patient remained intractably hypotensive so eventually the resuscitation was discontinued. The patient died 5 hours after her transfer to the intensive care unit.

At autopsy there was evidence of peritonitis with areas of intraperitoneal hemorrhage and traces of remaining food particles. The duodenum was markedly dilated and the small and large bowel were both congested. There was mild fatty change in the liver. The lungs were edematous and contained intrapulmonary hemorrhages. The renal parenchyma was congested with fibrin thrombi. Examination of the brain showed dilated lateral ventricles containing slightly blood-stained cerebrospinal fluid. These latter three findings all correlated pathologically with DIC.

The resected, decompressed stomach measured 41 cm along its greater curvature, 21 cm along its lesser curvature and 15 cm in diameter. The external surface of the stomach was bluish-black. There was marked thinning of the gastric wall with possible necrosis in several areas. A perforation 2 cm long was found on the lesser curvature near the gastroesophageal junction. This perforation had irregular, thinned-out edges that were hemorrhagic and appeared necrotic.

Because of the unusual presentation and surgical findings in this patient, a diagnosis of bulimia was suspected intraoperatively. An examination of the patient in the ICU revealed no dorsal scarring of the fingers or poor dentition as may be found with habitual purging. The only positive information came from questioning the patient's husband and mother-in-law. Although the husband was curious to know if this tragedy could have been caused by bulimia, he denied any knowledge of bulimic behaviour in his wife. However, the mother-in-law provided a much more consistent history. She gave details of massive grocery bills and remarked on the patient's ability to consume extraordinary amounts of food without gaining weight. The patient was never seen purging but always excused herself to the washroom immediately after one of her large meals. The patient was preoccupied with her self-image and exercised several hours a day. Interestingly, the mother-in-law also noted that the patient had swollen cheeks several weeks before presentation. This is observed when parotid gland hypertrophy develops, a problem not uncommon to bulimic patients.[1]

Discussion

Bulimia nervosa is a psychiatric eating disorder that has only been defined relatively recently.[2,3] Originally considered a variant of anorexia, it is now considered a separate entity with its own symptoms, signs, epidemiologic characteristics and psychopathologic features. The main difference between bulimia and anorexia is the occurrence of binge eating in bulimic patients, and it is this symptom that gives rise to most of the numerous medical complications of bulimia that have been documented since this disease was recognized. These complications include gastrointestinal, cardiovascular, endocrine and fluid and electrolyte disturbances.[4,5]

Acute gastric dilatation and perforation are relatively rare complications of bulimia, but they can have devastating consequences. Acute dilatation of the stomach can occur after the rapid ingestion of large quantities of food, since gastric emptying and esophageal motility are impaired in bulimic patients[6] as they are in anorexic patients.[7–9] Perforation occurs when gastric wall necrosis develops secondary to ischemia, when the wall tension exceeds the venous pres-

sure.[10] It has been suggested that gastric necrosis may not occur as a result of arterial insufficiency alone because there is extensive collateral circulation in the stomach.[11]

Gastric dilatation and perforation have been documented repeatedly in the literature, but there are very few reported cases related to eating disorders. The first case of acute dilatation in an anorexic person was published by Russell[12] in 1966, and the first case of perforation, also in an anorexic person, was reported by Evans[13] in 1968. The first death resulting from gastric perforation was reported by Lebriquir and associates[14] in 1978, again involving an anorexic woman. They described a second case in which a young anorexic woman died of a cardiac arrest during an attempt to decompress her acutely dilated stomach with a trocar. Saul, Dekker and Watson[15] in 1981 incorrectly claimed to report the first death from gastric perforation in an anorexic patient. By current definition the patients described by Russell and Saul, Dekker and Watson would likely be classified as bulimic. From a review of the literature we found three reports of gastric necrosis[16–18] and four more cases of gastric perforation[18–21] involving bulimic patients.

Of the eight patients (including ours) with gastric perforation, five presented with acute perforation, and two[14,21] presented with acute dilatation, which progressed to perforation on the 5th and 6th hospital days respectively. In the original report of perforation,[13] the stomach perforated intraoperatively after the patient presented with abdominal distension. Two[13,21] of the three patients who had perforation while in hospital survived, the third died.[14] Of the five patients who presented with perforation, only one survived.[19] The cause of death in the fatal cases reported prior to ours was either infarcted bowel or intractable hypotension. In none of these cases was DIC documented (Table I).

Our case was unusual in several respects. First, the patient was considerably older (43 years) than other patients who sustained a perforation (average 25.4 years).[19] Her symptoms developed much more precipitously than symptoms in all the other reports, in which the patients were either ambulatory or had already sought medical attention within the previous 24 hours. This is the first case in which DIC was documented in association with gastric perforation in a bulimic patient.

Table I. Reported Cases of Gastric Perforation in Women With Eating Disorders

Series	Age, yr	Diagnosis	Treatment	Outcome
Evans, 1968[13]	20	AN	Gastrectomy	Recovered
Lebriquir and associates, 1978[14]	20	AN	Gastrectomy	Died of septic shock
Saul, Dekker and Watson, 1981[15]	22	AN?	Laparotomy	Died of infarcted bowel
Abdu, Garritano and Culver, 1987[18]	17	BL	Gastrectomy	Died of shock
Breslow, Yates and Shisslak, 1986[19]	32	BL	Gastrectomy	Recovered
Jacquot and associates, 1989[20]	24	BL	Laparotomy	Died of hypotension
Petrin and associates, 1990[21]	25	?	Jejunostomy, NGT, abdominal drain Gastrectomy, splenectomy, Roux-en-Y anastomosis	Recovered
Present series, 1992	43	BL	Gastrectomy, Roux-en-Y anastomosis	Died of DIC

AN = anorexia nervosa, BL = bulimia, NGT = nasogastric tube, DIC = disseminated intravascular coagulation

Conclusions

Gastric dilatation, infarction and perforation can occur in either anorexic or bulimic patients. This is not unexpected because gastric motility dysfunction has been documented in both these conditions. Patients who present with gastric dilatation or necrosis but not perforation seem more likely to have a successful outcome (only two deaths[14,22] out of 15 cases[14–18,22]), whereas those who present with perforation have an extremely high mortality (four out of five cases). Patients who were already in hospital at the time of perforation were more likely to do well. This difference may relate to the duration of the peritonitis that results from perforation. Early medical attention is necessary to avoid deaths, and patients with eating disorders (or motility disorders) should be warned to be extremely wary of the onset of abdominal pain. As our case illustrates, bulimia can have a subtle subclinical course and can occur in any age group. This gastric complication of bulimia appears to be confined exclusively to women. It is anticipated that in the future this rare complication of bulimia will be preventable.

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Use of a Pericardial Xenograft Patch in Repair of Resected Retrohepatic Vena Cava

Carlos Del Campo, MD, FRCSC;* George P. Konok, MD, FRCSC†

Complete surgical excision of malignant tissue is mandatory for the long-term survival of patients with hepatic metastases from colorectal carcinoma. Involvement of the inferior vena cava (IVC) may prevent this resection. The authors describe the case of a 64-year-old man who underwent right hepatic lobectomy for metastases from colorectal carcinoma. Partial involvement of the IVC by tumour necessitated resection of a large portion of its wall. Venous continuity was re-established with a large bovine pericardial patch. At follow-up 2 years after the repair, the IVC was still patent and the vessel diameter was normal. On enhanced computed tomography, the patch was indistinguishable from the native vessel.

L'excision chirurgicale complète du tissu malin est essentielle à la survie prolongée des patients souffrant de métastases hépatiques d'un cancer colorectal. Une atteinte de la veine cave inférieure (VCI) peut empêcher cette résection. Les auteurs décrivent le cas d'un homme de 64 ans qui subit une lobectomie hépatique droite pour métastases d'un cancer colorectal. Une atteinte partielle de la VCI nécessita la résection d'une large partie de la paroi. La circulation veineuse fut rétablie à l'aide d'une large pièce de tissu péricardique d'origine bovine. À l'examen de contrôle, 2 ans après la réparation, la VCI était toujours perméable et le vaisseau était de diamètre normal. À la tomographie par ordinateur, la pièce ne pouvait être distinguée du vaisseau lui-même.

Hepatic resection is the treatment of choice for patients with liver metastases from colorectal carcinoma. Complete surgical excision of malignant tissue is critical for long-term survival. Unresectable lesions carry a poor prognosis: a median survival of 15 months and 21 months for multiple and solitary metastases respectively.[1] Patients who undergo resection have a 5-year survival rate ranging from 20% to 45%.[2] Resection is contraindicated if there is tumour recurrence at the primary site or if there are distant metastases and may be impossible, due to anatomic limitations, if there is direct invasion of the inferior vena cava (IVC) by tumour. We describe a patient in whom such a situation necessitated resection and repair of the IVC to achieve complete removal of visible tumour.

Case Report

A 64-year-old man was referred to the surgical gastroenterology service of our institution for evaluation of hepatic metastases. He had undergone resection 1 year earlier for adenocarcinoma of the sigmoid colon. Investigation for metastases at the time of surgery and follow-up colonoscopy had given negative results. However, an increase in the carcinoembryonic antigen levels had prompted computed tomography (CT) of the abdomen, which had demonstrated four lesions in the right lobe of the liver but none in the left lobe. The largest of these masses appeared to be encroaching on the retrohepatic vena cava. Abdominal ultrasonography confirmed the presence of the four masses. The diameter of the IVC appeared to be normal, without extension of tumour into its lumen. Angiography failed to demonstrate tumour involvement of the IVC wall. Resection of the tumour without involvement of the IVC was judged to be feasible.

A right hepatic lobectomy was attempted. Exploration of the retrohepatic vena cava revealed tumour extension into its wall. It became obvious that to achieve complete resection of the tumour a portion of the IVC would have to be resected. Intra-abdominal ultrasonography was not used, because of its inability to rule out tumour invasion of the

*From the *Division of Vascular Surgery and the †Division of General Surgery, Camp Hill Medical Centre, and Dalhousie University, Halifax, NS*

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Reprint requests to: Dr. Carlos Del Campo, 100 East Valencia Mesa Dr., Suite 301, Fullerton, CA 92635, USA

vessel wall. A vascular surgeon was consulted intraoperatively. The IVC was cross-clamped at the level of the diaphragm and 2 cm below the tumour. The portal vein was also clamped. A 5 × 4-cm segment of the IVC was removed (Fig. 1). A patch of the same dimensions was tailored from a sheath of formaldehyde-preserved bovine pericardium (Shiley Laboratories, Irvine, Calif.). The patch was sutured with its epicardial side toward the lumen and covered over two-thirds of the inferior vena caval circumference (Fig. 2). The cross-clamp time was 17 minutes. The patient's mean systolic arterial pressure was maintained at 80 mm Hg during venous clamping.

The patient tolerated the procedure well and was discharged from hospital 2 weeks after the operation. He was asymptomatic at the time of discharge. CT 2 years later showed a patent IVC patch (Fig. 3) with a normal vessel diameter. There were two small metastatic lesions in one lung. Thirty-four months after resection he was still asymptomatic. There was no evidence of venous hypertension of the lower extremities.

Discussion

Extension of renal cell carcinoma into the IVC is currently managed by nephrectomy and tumour thrombectomy. Extended survival has been achieved after complete surgical extirpation.[3,4] Reports of similar resections for non-renal malignant tumours have been scant. A Medline literature computer search complemented by a manual search of the *Index Medicus* and the *Yearbook of Vascular Surgery* did not reveal any reports of retrohepatic caval resection for colorectal metastases. In cases of renal cell carcinoma with direct, major invasion of the vena caval wall, resection of the involved segment has occasionally been performed. Caval continuity is restored preferably with synthetic polytetrafluoroethylene (PTFE) interposition grafts.[5] Autogenous grafts have had limited clinical use because they are not available in sufficient length or

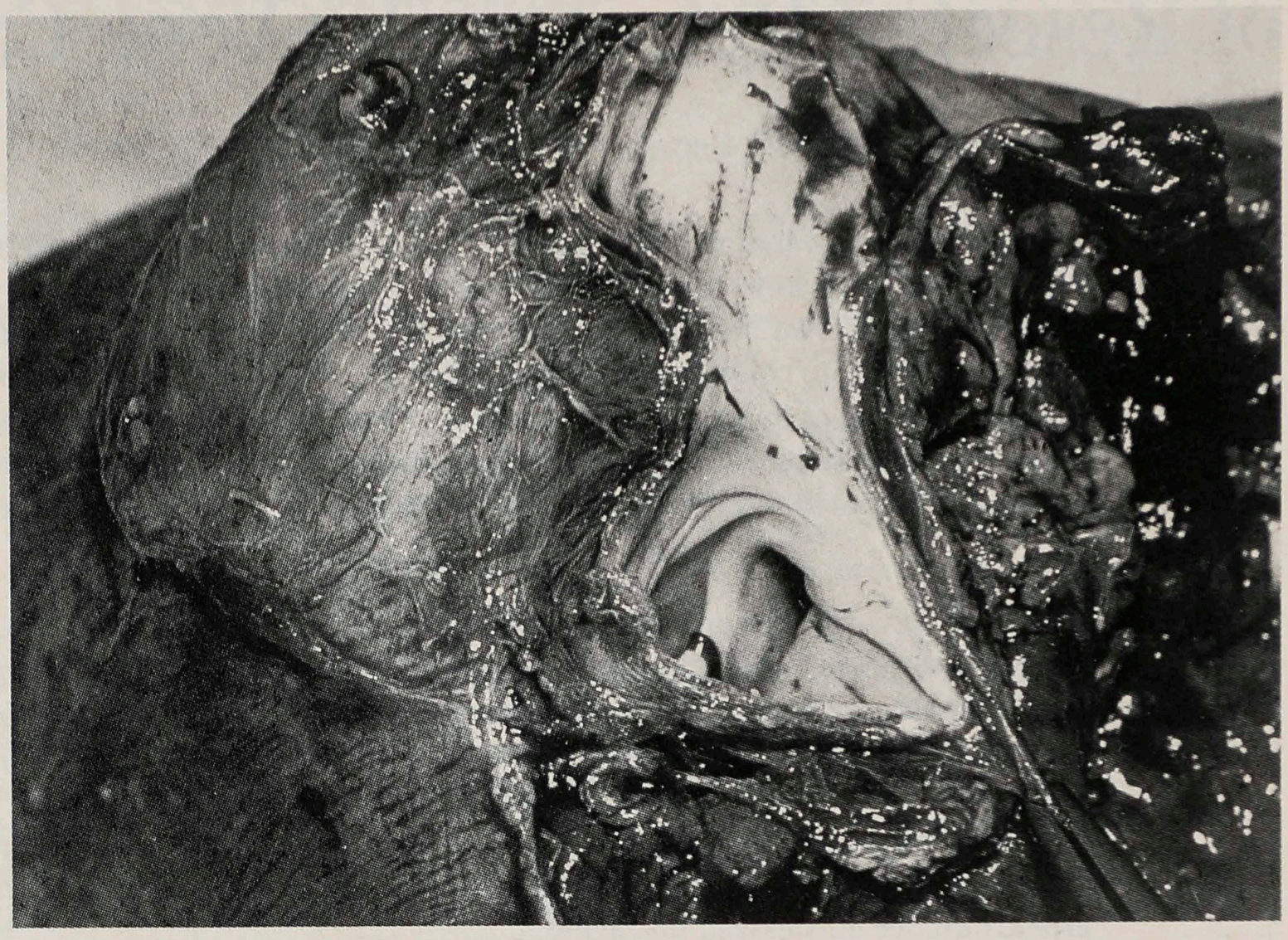

FIG. 1. Operative specimen showing extensive resection of inferior vena cava (IVC). Note orifices of right hepatic veins draining into IVC. Tumour encases IVC but does not invade intimal layer.

FIG. 2. Pericardial xenograft patch can be seen in centre below surface of liver. Repair conforms well to caval diameter without kinking and extends superiorly to level of diaphragm.

width. The ideal venous substitute (as outlined by Walter[6]) has not yet been described.

In our patient, preoperative ultrasonography, CT and arteriography demonstrated lack of intraluminal involvement of the IVC. Intra-abdominal ultrasonography was judged unnecessary because it would not categorically rule out tumour cell invasion into the wall of the IVC. On direct visualization after dissection, it appeared that the tumour had invaded the wall of the IVC. The inferior vena caval wall invasion was confirmed by examination of the resected specimen. The intima appeared to be intact (Fig. 1). Both findings were further confirmed by histopathologic examination. Our patient required resection of two-thirds of the total caval circumference to achieve satisfactory margins of resection. If the IVC had been replaced by an interposition graft, the left hepatic veins would have required reimplantation onto the graft. For this reason we could not use the spiral vein graft popularized by Doty.[7] Thus, the reconstruction of choice consisted of a large patch to restore caval diameter.

Clinical and experimental work has shown that, when used to replace veins, PTFE grafts have a high incidence of thrombosis.[8] We have demonstrated experimentally that glutaraldehyde-fixed formaldehyde-preserved bovine pericardium has a patency rate of 80% at a mean follow-up of 13 ± 8 months when used as an interposition graft in the superior vena cava.[9] Intraoperative evaluation showed that a pericardial patch would conform much better than a PTFE graft and would provide consistency and compliance that more closely resembled the native IVC. Microscopic studies in our experimental model showed focal calcification in both pericardial and PTFE grafts, but the calcification did not influence graft performance.[9] CT performed in our patient 2 years postoperatively did not show any evidence of calcification. Furthermore, there was no difference radiologically between the patch and the native IVC. During resection, temporary intraluminal shunting was not used, because a trial of fluid loading and IVC occlusion was well tolerated.

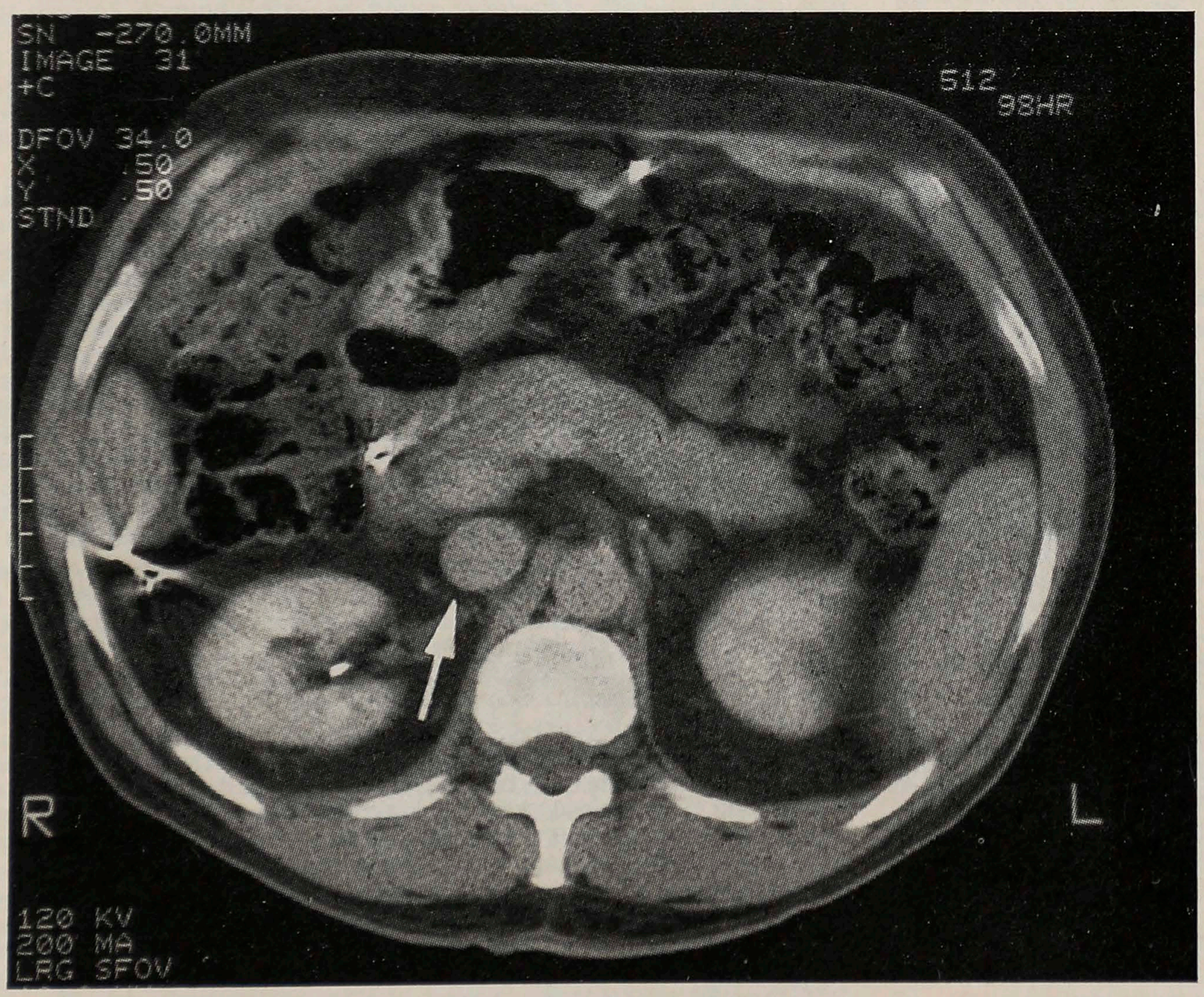

FIG. 3. Computed tomography scan 2 years after repair. IVC is patent (arrow). There is no evidence of calcification.

Conclusions

A bovine pericardial patch provided satisfactory hemodynamic and anatomic performance when used for reconstruction of the IVC in a patient who underwent hepatic resection of metastatic tumour from colorectal cancer. Larger clinical series are necessary before this procedure can be considered for routine application.

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TORADOL®

10 mg tablets & 30 mg/mL IM injections KETOROLAC TROMETHAMINE

Effectively treating acute pain

TORADOL® (ketorolac tromethamine) 10 mg tablets
TORADOL® IM (ketorolac tromethamine injection) 10 mg/mL, 15 mg/mL or 30 mg/mL intramuscular injection
THERAPEUTIC CLASSIFICATION: Analgesic Agent

ACTION:
Toradol (ketorolac tromethamine) is a non-steroidal anti-inflammatory drug (NSAID) that exhibits analgesic activity mediated by peripheral effects. Ketorolac inhibits the synthesis of prostaglandins through inhibition of the cyclo-oxygenase enzyme system. At analgesic doses, it has minimal anti-inflammatory and antipyretic activity. Pain relief is comparable following the administration of ketorolac by intramuscular or oral routes. The peak analgesic effect occurs at 2-3 hours post-dosing with no evidence of a statistically significant difference over the recommended dosage range. The greatest difference between large and small doses of Toradol administered by either route is in the duration of analgesia. Ketorolac tromethamine is rapidly and completely absorbed when administered by either the oral or the intramuscular route. The pharmacokinetics are linear following single and multiple dosing. Steady state plasma levels are attained after one day of Q.I.D. dosing. Following oral administration, peak plasma concentrations of 0.7 to 1.1 µg/mL occurred at an average of 44 minutes after a single 10 mg dose. The terminal plasma elimination half-life ranged between 2.4 and 9.0 hours in healthy adults, while in elderly subjects (mean age = 72 years), it ranged between 4.3 and 7.6 hours. A high fat meal decreased the rate, but not the extent, of absorption of oral ketorolac tromethamine.

The use of an antacid had no effect on the pharmacokinetics of ketorolac. Following intramuscular administration, peak plasma concentrations of 2.2 to 3.0 µg/mL occurred an average of 50 minutes after a single 30 mg dose. The terminal plasma half-life ranged between 3.5 and 9.2 hours in young adults and between 4.7 and 8.6 hours in elderly subjects (mean age = 72 years). In renally impaired patients there is a reduction in clearance and an increase in the terminal half-life of ketorolac tromethamine (see table below). The primary route of excretion of ketorolac tromethamine and its metabolites (conjugates and the p-hydroxy metabolite) is in the urine (91.4%) with the remainder (6.1%) being excreted in the feces. More than 99% of the ketorolac in plasma is protein bound over a wide concentration range. The hemodynamics of anaesthetized patients were not altered by parenteral administration of Toradol.

THE INFLUENCE OF AGE, LIVER AND KIDNEY FUNCTION ON THE CLEARANCE AND TERMINAL HALF-LIFE OF TORADOL IM[1] AND ORAL[2]

TYPES OF SUBJECTS	TOTAL CLEARANCE (in L/h/kg)[3]		TERMINAL HALF-LIFE (in hours)	
	IM MEAN (range)	ORAL MEAN (range)	IM MEAN (range)	ORAL MEAN (range)
Normal Subjects IM (n=54) Oral (n=77)	0.023 (0.010-0.046)	0.025 (0.013-0.050)	5.3 (3.5-9.2)	5.3 (2.4-9.0)
Healthy Elderly Subjects IM (n=13), Oral (n=12) (mean age = 72, range = 65-78)	0.019 (0.013-0.034)	0.024 (0.018-0.034)	7.0 (4.7-8.6)	6.1 (4.3-7.6)
Patients with Hepatic Dysfunction IM and Oral (n=7)	0.029 (0.013-0.066)	0.033 (0.019-0.051)	5.4 (2.2-6.9)	4.5 (1.6-7.6)
Patients with Renal Impairment IM and Oral (n=9)(serum creatinine 1.9 - 5.0 mg/dL)	0.014 (0.007-0.043)	0.016 (0.007-0.052)	10.3 (8.1-15.7)	10.8 (3.4-18.9)
Renal Dialysis Patients IM (n=9)	0.016 (0.003-0.036)		13.6 (8.0-39.1)	

[1] Estimated from 30 mg single IM doses of ketorolac tromethamine
[2] Estimated from 10 mg single oral doses of ketorolac tromethamine
[3] Litres/hour/kilogram

INDICATIONS:
Oral Route: Orally administered Toradol (ketorolac tromethamine) is indicated for the short-term management of mild to moderately severe pain, including post-surgical pain (such as general, orthopaedic and dental surgery), acute musculoskeletal trauma pain and post-partum uterine cramping pain.

Intramuscular Route: Intramuscular injection of Toradol is indicated for the short-term management of moderate to severe pain, including pain following major abdominal, orthopaedic and gynecological operative procedures.

CONTRAINDICATIONS:
Hypersensitivity: Like other non-steroidal anti-inflammatory drugs, Toradol (ketorolac tromethamine) has been associated with hypersensitivity reactions. Toradol should not be used when there is a known or suspected hypersensitivity to the drug. Because of the possibility of cross-sensitivity, Toradol should not be used in patients with the complete or partial syndrome of nasal polyps, angioedema, bronchospastic reactivity (e.g. asthma) or other allergic manifestations to acetylsalicylic acid (ASA) or other non-steroidal anti-inflammatory drugs. Severe and fatal anaphylactoid reactions have occurred in such individuals.

Gastrointestinal: As with other NSAIDs, Toradol also should not be used in patients with peptic ulcer or active inflammatory disease of the gastrointestinal system. Severe and fatal reactions have occurred in such individuals.

WARNINGS:
The long-term administration of Toradol (ketorolac tromethamine) is not recommended. The most serious risks associated with NSAIDs including Toradol are:

Gastrointestinal Ulcerations, Bleeding and Perforation: Serious gastrointestinal toxicity, such as bleeding, ulceration, and perforation, can occur at any time, with or without warning symptoms, during therapy with non-steroidal anti-inflammatory drugs. To date, studies with NSAIDs have not identified any subset of patients not at risk for developing peptic ulceration and bleeding. Post-marketing experience with Toradol suggests that there may be a greater risk of gastrointestinal ulcerations, bleeding, and perforation in the elderly, and most spontaneous reports of fatal gastrointestinal events are in the aged population.

LONG-TERM USE OF TORADOL: The oral use of Toradol 10 mg QID on a long-term basis is associated with more gastrointestinal tract adverse effects than is ASA 650 mg QID.
In a clinical trial in 823 patients with chronic pain states comparing Toradol tablets 10 mg QID (553 patients) with ASA 650 mg QID (270 patients), during the first week there was a 2.4% dropout rate because of upper GI complaints in the Toradol tablets treated patients compared with 0.4% rate in the ASA treated group. After the first 2 weeks, the dropout rate due to GI pain or discomfort were comparable in both treatment groups. The time-adjusted percentages, which are not statistically significantly different, of patients who developed ulcer or upper GI bleeding are as follows:

	CUMULATIVE OCCURRENCE	
INTERVAL	KETOROLAC	ASA
≤ 3 months	0.69*	0*
≤ 6 months	1.59*	0.73*

*There was no statistically significant difference between ketorolac and ASA at either of the intervals tested.

PHYSICIANS SHOULD CAREFULLY WEIGH THE POTENTIAL RISKS AND BENEFITS OF USING TORADOL TABLETS ON A LONG-TERM BASIS. PATIENTS SHOULD BE INSTRUCTED TO WATCH FOR SIGNS OF SERIOUS GI ADVERSE EVENTS AND THEY SHOULD BE MONITORED MORE CLOSELY THAN IF THEY WERE ON ANOTHER NSAID.

Renal Toxicity: The following renal abnormalities have been associated with Toradol and other drugs that inhibit renal prostaglandin biosynthesis: acute renal failure, nephrotic syndrome, interstitial nephritis, renal papillary necrosis.

Haemorrhage: Postoperative haematomas and other symptoms of wound bleeding have been reported in association with the perioperative use of intramuscular Toradol. If Toradol is to be administered to patients who have coagulation disorders or who are receiving drug therapy that interferes with haemostasis, careful observation is advised.

Hypersensitivity Reactions: The possibility of severe or fatal hypersensitivity reactions should be considered, even for patients with no known history of previous exposure or hypersensitivity to Toradol or other NSAIDs. As with other NSAIDs, patients should be questioned for history of allergy to NSAIDs or ASA or for the syndrome consisting of nasal polyps, ASA allergy and asthma before being prescribed Toradol. Asthmatic patients with triad asthma (the syndrome of nasal polyps, asthma and hypersensitivity to ASA or other NSAIDs) may be at particular risk for severe hypersensitivity reactions.

Other NSAIDs: Ketorolac tromethamine is not recommended for concurrent use with other NSAIDs because of the potential for additive side effects.

Use in Pregnancy and Lactation: The administration of ketorolac tromethamine is not recommended during pregnancy or lactation. After 1 day at 10 mg QID. oral dosing, Toradol has been detected in the milk of lactating women at a maximum concentration of 7.9 ng/mL.

Use in Labour: Ketorolac tromethamine is not recommended for use as an obstetrical preoperative medication or for obstetrical analgesia because of the known effects of NSAIDs on uterine contraction and fetal circulation.

Use in Children: Safety and efficacy in children have not been established. Therefore, Toradol is not recommended for use in children under age 16.

Use in the Elderly: Because ketorolac is cleared somewhat more slowly by the elderly (See PHARMACOKINETICS) who are also more sensitive to the gastrointestinal and renal effects of NSAIDs (See WARNINGS and PRECAUTIONS), extra caution and the lowest effective dose (See DOSAGE AND ADMINISTRATION) should be used.

PRECAUTIONS:
Physicians should be alert to the pharmacologic similarity of Toradol (ketorolac tromethamine) to other non-steroidal anti-inflammatory drugs that inhibit cyclo-oxygenase. Toradol is not an anesthetic agent and possesses no sedative or anxiolytic properties.

Gastrointestinal Effects: Close medical supervision is recommended in patients prone to gastrointestinal tract irritation, particularly those with a history of peptic ulcer, diverticulosis or other inflammatory disease of the gastrointestinal tract. In these cases, the physician must weigh the benefits of treatment against the possible hazards. Patients taking any NSAID including ketorolac tromethamine should be instructed to contact a physician immediately if they experience symptoms or signs suggestive of peptic ulceration or gastrointestinal bleeding. These reactions can occur at any time during the treatment. If peptic ulceration is suspected or confirmed, or if gastrointestinal bleeding occurs, ketorolac tromethamine should be discontinued and appropriate treatment instituted with close patient monitoring.

Renal Effects: As with other drugs that inhibit prostaglandin biosynthesis, elevations of blood urea nitrogen (BUN) and creatinine have been reported in clinical trials with (ketorolac tromethamine). Since ketorolac tromethamine and its metabolites are excreted primarily by the kidney, the following precautions are indicated for patients with: **Severely impaired renal function** (serum creatinine values greater than 5 mg/dL, 442 µmol/L): Toradol is not recommended; **Moderately impaired renal function** (serum creatinine values ranging from 1.9 to 5.0 mg/dL, 168 to 442 µmol/L) - The total daily dose of ketorolac tromethamine should be reduced by half. In these patients the rate of ketorolac tromethamine clearance was reduced to approximately half of normal. Patients who are volume depleted may be dependent on renal prostaglandin production to maintain renal perfusion and, therefore, glomerular filtration rate. In such patients, the use of drugs which inhibit prostaglandin synthesis has been associated with further decreases in renal blood flow. Predisposing factors include sepsis, impaired renal function, heart failure, liver dysfunction, diuretic therapy, and advanced age. Caution is advised if ketorolac tromethamine is used in such circumstances. Close monitoring of urine output, serum urea and serum creatinine is recommended until renal function recovers.

Hepatic Effects: Meaningful elevations (greater than 3 times normal) of serumtransaminases (glutamate pyruvate (SGPT or ALT) and glutamic oxalacetic (SGOT or AST)), occurred in controlled clinical trials in less than 1% of patients. If clinical signs and symptoms consistent with liver disease develop, or if systemic manifestations occur (e.g., eosinophilia, rash, etc.), ketorolac tromethamine should be discontinued. Patients with impaired hepatic function from cirrhosis do not have any clinically important changes in ketorolac tromethamine clearance. Studies in patients with active hepatitis or cholestasis have not been performed.

Fluid and Electrolyte Balance: Fluid retention and edema have been observed in patients treated with Toradol. Therefore, as with many other NSAIDs, the possibility of precipitating congestive heart failure in elderly patients or those with compromised cardiac function should be considered. Toradol should be used with caution in patients with cardiac decompensation, hypertension or other conditions which cause a predisposition to fluid retention.

Hematologic Effects: Ketorolac tromethamine inhibits platelet function and may prolong bleeding time. It does not affect platelet count, prothrombin time (PT) or partial thromboplastin time (PTT). Unlike the prolonged effects from ASA the inhibition of platelet function by ketorolac tromethamine is normalized within 24 to 48 hours after the drug is discontinued. Patients on full anti-coagulation therapy (e.g. heparin or dicumarol derivatives) may be at increased risk of bleeding if given Toradol concurrently. Thus, the benefit should be weighed against this risk. The concomitant use of Toradol and heparin (5000 U s.c. BID) appears to be associated with less risk (see DRUG INTERACTIONS). In patients receiving anticoagulants, the risk of intramuscular haematoma formation from Toradol IM injections may be increased. In post-marketing experience, postoperative wound haemorrhage has been reported with the use of Toradol. Therefore, caution should be exercised when strict haemostasis is critical. Toradol IM is

ecommended as a pre-operative or intra-operative medication because of the risk of ssive bleeding. Blood dyscrasias associated with the use of NSAIDs are rare, could occur with severe consequences.

ction: In common with other non-steroidal anti-inflammatory drugs, ketorolac ethamine may mask the usual signs of infection.

G INTERACTIONS:

ein Binding: Toradol (ketorolac tromethamine) is highly bound to human plasma ein (mean 99.2%) and binding is independent of concentration. As ketorolac ethamine is a highly potent drug and present in low concentrations in plasma, it would not xpected to displace other protein-bound drugs significantly. Therapeutic concentrations goxin, warfarin, acetaminophen, phenytoin, and tolbutamide did not alter ketorolac ethamine protein binding.

coagulant Therapy: Prothrombin time should be carefully monitored in all patients iving oral anticoagulant therapy concomitantly with ketorolac tromethamine. Toradol IM n with two doses of 5000 U of heparin to 11 healthy volunteers resulted in a mean tem- bleeding time of 6.4 min (3.2-11.4 min) compared to a mean of 6.0 min (3.4-7.5 min) for arin alone and 5.1 min (3.5-8.5 min) for placebo. The *in vitro* binding of warfarin to plasma eins is only slightly reduced by ketorolac tromethamine (99.5% control vs. 99.3%) lasma concentrations of 5 to 10 µg/mL.

oxin: Ketorolac tromethamine does not alter digoxin protein binding.

cylates: *In vitro* studies indicated that, at therapeutic concentrations of salicylates µg/mL), the binding of ketorolac tromethamine was reduced from approximately 99.2% to % representing a potential two-fold increase in unbound Toradol plasma levels.

yme Induction: There is no evidence, in animal or human studies, that ketorolac ethamine induces or inhibits the hepatic enzymes capable of metabolizing itself or other s. Hence, it would not be expected to alter the pharmacokinetics of other drugs due to yme induction or inhibition mechanisms.

benecid: Concomitant administration of ketorolac tromethamine and probenecid lts in the decreased clearance of ketorolac and a significant increase in ketorolac plasma ls (approximately three-fold increase) and terminal half-life (approximately two-fold ease).

osemide: Ketorolac tromethamine reduces the diuretic response to furosemide approximately 20% in normovolemic subjects.

ium: Some NSAIDs have been reported to inhibit renal lithium clearance, leading to increase in plasma lithium concentrations and potential lithium toxicity. The effect of rolac tromethamine on lithium plasma levels has not been studied.

hotrexate: The concomitant administration of methotrexate and some NSAIDs has en reported to reduce the clearance of methotrexate, thus enhancing its toxicity. The ct of ketorolac tromethamine on methotrexate clearance has not been studied.

phine: Intramuscular Toradol has been administered concurrently with morphine veveral clinical trials of postoperative pain without evidence of adverse interactions.

VERSE EVENTS:

ADOL TABLETS: Short-Term Patient Studies - The incidence of adverse reactions in 371 ients receiving multiple 10 mg doses of Toradol (ketorolac tromethamine) for pain resulting n surgery or dental extraction during the post-operative period (less than 2 weeks) is listed ow. These reactions may or may not be drug related. **Incidence between 4 and 9%: vous system -** somnolence, insomnia; **Digestive system -** nausea. **Incidence between 2 and Nervous system -** nervousness, headache, dizziness; **Digestive system -** diarrhea, dyspep- gastrointestinal pain, constipation. **Body as a whole -** fever. **Incidence 1% or Less: Nervous em -** abnormal dreams, anxiety, dry mouth, hyperkinesia, paresthesia, increased sweating, phoria, hallucinations; **Digestive system -** anorexia, flatulence, vomiting, stomatitis, gastritis, strointestinal disorder, sore throat; **Body as a whole -** asthenia, pain, back pain; **rdiovascular system:** vasodilatation, palpitation, migraine, hypertension; **Respiratory system -** ugh increased, rhinitis, dry nose; **Musculo-skeletal system -** myalgia, arthralgia; **Skin and pendages -** rash, urticaria; **Special senses -** blurred vision, ear pain; **Urogenital system:** uria.

g-Term Patient Study - The adverse reactions listed below were reported to be probably ated to study drug in 553 patients receiving long-term oral therapy (approximately 1 year) n Toradol. **Incidence between 10 and 12%: Digestive system -** dyspepsia, gastrointestinal n.

idence Between 4 and 9%: Digestive system - nausea, constipation; **Nervous system -** adache. **Incidence Between 2 and 3%: Digestive system -** diarrhea, flatulence, gastrointesti- fullness, peptic ulcers; **Nervous system -** Dizziness, somnolence; **Metabolic/Nutritional disor- -** edema. **Incidence 1% or Less: Digestive system -** eructation, stomatitis, vomiting, anorex- duodenal ulcer, gastritis, gastrointestinal haemorrhage, increased appetite, melena, mouth eration, rectal bleeding, sore mouth; **Nervous system -** abnormal dreams, anxiety, depres- n, dry mouth, insomnia, nervousness, paresthesia; **Special senses -** tinnitus, taste perversion, normal vision, blurred vision, deafness, lacrimation disorder; **Metabolic/Nutritional disorder -** eight gain, alkaline phosphatase increase, BUN increased, excessive thirst, generalized ema, hyperuricemia; **Skin and appendages -** pruritus, rash, burning sensation skin; **Body as whole -** asthenia, pain, back pain, face edema, hernia; **Musculo-skeletal system -** arthral- , myalgia, joint disorder; **Cardiovascular system:** chest pain, chest pain substernal, graine; **Respiratory system -** dyspnea, asthma, epistaxis; **Urogenital system -** hematuria, reased urinary frequency, oliguria, polyuria; **Hemic and lymphatic -** Anemia, purpura.

RADOL IM: The adverse reactions listed below were reported in Toradol IM clinical effica- trials. In these trials patients (n=660) received either single 30 mg doses (n=151) or multiple mg doses (n=509) over a time period of 5 days or less for pain resulting from surgery.

ese reactions may or may not be drug related. **Incidence Between 10 and 13%: Nervous stem -** somnolence; **Digestive system -** Nausea. **Incidence Between 4 and 9%: Nervous sys- m -** headache; **Digestive system -** vomiting; **Injection site -** injection site pain. **Incidence tween 2 and 3%: Nervous System -** sweating, dizziness; **Cardiovascular system -** vasodilata- n. **Incidence 1% or Less: Nervous system -** insomnia, increased dry mouth, abnormal eams, anxiety, depression, paraesthesia, nervousness, paranoid reaction, speech disorder, phoria, libido increased, excessive thirst, inability to concentrate, stimulation; **Digestive sys- m -** flatulence, anorexia, constipation, diarrhea, dyspepsia, gastrointestinal fullness, gastroin- stinal haemorrhage, gastrointestinal pain, melena, sore throat, liver function abnormalities, ctal bleeding, stomatitis; **Cardiovascular system -** hypertension, chest pain, tachycardia, emorrhage, palpitation, pulmonary embolus, syncope, ventricular tachycardia, pallor, flush- g; **Injection site -** injection site reaction; **Body as a whole -** asthenia, fever, back pain, chills, ain, neck pain; **Special senses -** taste perversion, tinnitus, blurred vision, diplopia, retinal emorrhage; **Musco-skeletal system -** myalgia, twitching; **Respiratory system -** asthma, ough increased, dyspnea, epistaxis, hiccup, rhinitis; **Skin and appendages -** pruritus, rash, sub- taneous hematoma, skin disorder; **Urogenital system -** dysuria, urinary retention, olliguria, creased urinary frequency, vaginitis; **Metabolic/nutritional disorders -** edema, hypokalemia, povolemia **Hemic and lymphatic system -** anemia, coagulation disorder, purpura.

st-Marketing Experience: The following post-marketing adverse experiences, although re (1% or less), have been reported for patients who have received either formulation of Toradol.

Renal events - acute renal failure, flank pain with or without haematuria and/or azotemia; **Hypersensitivity reactions:** bronchospasm, laryngeal edema, hypotension, flushing, rash, and anaphylactoid reactions, such reactions have occurred in patients with no prior history of hypersensitivity. **Gastrointestinal events -** gastrointestinal hemorrhage, peptic ulceration, gastrointestinal perforation; **Hematologic events -** postoperative wound haemorrhage, rarely requiring blood transfusion (see PRECAUTIONS), thrombocytopenia; **Central nervous system-** convulsions, abnormal dreams, hallucinations, hyperkinesia, hearing loss; **Cardiovascular -** pulmonary edema; **Dermatology -** Lyell's syndrome, Stevens - Johnson syndrome, exfoliative dermatitis, maculopapular rash.

OVERDOSAGE: The absence of experience with acute overdosage precludes characterization of sequelae and assessment of antidotal efficacy at this time. In a gastroscopic study of healthy subjects, daily doses of 360 mg given over an 8-hour interval for each of five consecutive days (3 times the highest recommended dose) caused pain and peptic ulcers which resolved after discontinuation of dosing.

DOSAGE AND ADMINISTRATION:

Adults: Dosage should be adjusted according to the severity of the pain and the response of the patient. **Oral**: The usual oral dose of Toradol (ketorolac tromethamine) is 10 mg every 4 to 6 hours for pain as required. Doses exceeding 40 mg per day are not recommended. Toradol is recommended for short-term use only, i.e., for a maximum of a few weeks.

Parenteral: The recommended usual initial dose is 30 mg. Subsequent dosing may be 10 mg to 30 mg every 4-6 hours as needed to control pain. It is recommended that the administration of Toradol IM be limited to short-term therapy (not over 5 days) and the total daily dose should not exceed 120 mg.

This is because the risk of toxicity appears to increase with longer use at recommended doses (see WARNINGS and PRECAUTIONS). The administration of continuous multiple daily doses of Toradol IM has not been extensively studied. There has been limited experience with intramuscular dosing for more than 3 days since the vast majority of patients have transferred to oral medication or no longer required analgesic therapy after this time. In the initial post-operative period, more frequent dosing (e.g. every 2 hours) may be employed but the total daily dosing should not exceed 120 mg/day. If supplementary analgesia is required, a concomitant low dose of opiate can be used.

Patients under 50 kg, over age 65 years, or with less severe pain at baseline: the lower end of the dosage range (10-15 mg 4 times a day) is recommended.

Impaired Renal Function - Moderate - In patients with impaired renal function (serum creatinine values ranging from 1.9 to 5.0 mg/dL or 168 to 442 µmol/L), the total daily dose of Toradol should be reduced by half;

Severe - In patients with severely impaired renal function (serum creatinine values greater than 5 mg/dL, or 442 µmol/L) Toradol is not recommended.

Conversion from Parenteral to Oral Therapy: Toradol tablets may be used either as monotherapy or as follow-on therapy to parenteral ketorolac. Toradol IM should be replaced by an oral analgesic as soon as feasible. When Toradol tablets are used as a follow-on therapy to parenteral ketorolac, the total combined daily dose of ketorolac (oral + parenteral) should not exceed 120 mg on the day the change of formulation is made. Subsequent oral dosing should not exceed the recommended daily maximum of 40 mg.

Directions for Use of the Prefilled Syringes: Insert the plunger into the syringe barrel and thread it onto the screw. WITHOUT REMOVING THE NEEDLE GUARD, apply quick, firm pressure to the plunger to break the inner seal. (You will feel it let go). Pull back on the plunger slightly to relieve pressure. Remove the needle guard by twisting as you pull. Use the unit as you would a normal syringe. Dispose of properly. Single use only. Discard Unused Portions. Parenteral drug products should be inspected visually for particulate material and discoloration prior to use. Toradol (ketorolac tromethamine) is a Schedule F drug.

Stability and Storage Recommendations:

Toradol Tablets: Store at room temperature with protection from light.

Toradol IM: Store at room temperature with protection from light.

Availability of Dosage Forms: Toradol (ketorolac tromethamine) is available as 10 mg white round film coated tablets containing ketorolac tromethamine, microcrystalline cellulose, lactose and magnesium stearate with one side printed in red with TORADOL inside bold T and other side with Syntex. Toradol (ketorolac tromethamine) 10 mg tablets are available in bottles of 100 and 500 tablets.

Toradol IM is available in 1 mL ampoules (trays of 10) containing 10 or 30 mg/mL.
Toradol IM is also available in 1 mL syringes (1/box) containing 15 or 30 mg/mL.

Product Monograph available on request.

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Toradol Information Line: 1-800-561-5481.

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Pseudoaneurysm of the Cystic Artery: a Rare Cause of Hemobilia

Carlos A. Barba, MD;* Patrice M. Bret, MD;† E. John Hinchey, MD, MSc, FACS, FRCSC*

Aneurysms are a rare cause of hemobilia. The arteries most frequently involved are branches of the hepatic or gastroduodenal arteries. The authors report the case of a patient with hemobilia secondary to a pseudoaneurysm of the cystic artery. Only six other cases have been reported in the literature, and in all of them the condition was associated with inflammation of the gallbladder. Selective hepatic arteriography is the procedure of choice for diagnosis. Colour-Doppler ultrasonography aided in the diagnosis in the patient described in this report and may prove to be useful in equivocal cases. Cholecystectomy and ligation of the cystic artery with proximal control of the hepatic artery should be performed as soon as the diagnosis is made.

Les anévrismes sont une cause rare d'hémobilie. Les artères les plus fréquemment impliquées sont les troncs des artères hépatiques et gastroduodénales. Les auteurs présentent le cas d'un patient qui a souffert d'hémobilie secondaire à un pseudo-anévrisme de l'artère cystique. Seulement six autres cas ont été signalés dans la presse médicale et, dans tous les cas, l'affection était reliée à une inflammation de la vésicule biliaire. L'artériographie hépatique sélective est le meilleur moyen de diagnostic. Ici, l'échographie couleur de Doppler a aidé au diagnostic du patient; elle pourrait s'avérer utile dans les cas équivoques. Une cholécystectomie et une ligature de l'artère cystique avec un contrôle de l'artère hépatique devraient être pratiquées aussi tôt que le diagnostic est posé.

A rare cause of hemobilia is the erosion of intra- or extrahepatic bile ducts by arteriosclerotic, false or mycotic aneurysms of the hepatic or gastroduodenal artery.

We present a case of hemobilia caused by a pseudoaneurysm of the cystic artery, and we discuss the pertinent literature.

Case Report

A 70-year-old man was seen in the emergency department with a 3-day history of intermittent epigastric pain, which was constant and radiated to the right upper quadrant. He had also vomited a small amount of coffee grounds material on the day of presentation. The patient denied experiencing melena, fever or chills, any previous similar episode or any other gastrointestinal symptom. He had some tenderness in the right upper quadrant, and the stools and nasogastric tube drainage were positive for occult blood. On admission his hemoglobin level was 114 g/L, the hematocrit was 0.33, the leukocyte count was 12.9×10^9/L and the total serum bilirubin was 62 µmol/L (direct, 47 µmol/L); the serum electrolyte levels and serum amylase level were normal. He was not clinically icteric.

The patient was admitted for observation. On the 1st day in hospital his hemoglobin level dropped to 91 g/L. He remained hemodynamically stable, with a heart rate less than 100 beats/min. He did not have a postural drop in blood pressure. Gastroscopy revealed a polypoid mass with ulceration in the duodenal cap and a large diverticulum in the third stage of the duodenum, with a small amount of blood in the area. Abdominal ultrasonography showed one gallstone and a very thickened and distended gallbladder wall with heterogeneous contents. He was prescribed H_2 blockers for upper gastrointestinal bleeding and a presumed duodenal ulcer. Antibiotics (ampicillin, gentamicin and metronidazole) were started for a presumed diagnosis of chronic rather than acute cholecystitis.

The patient's condition remained relatively stable during the next few

*From the *Department of Surgery and the †Department of Radiology, McGill University, Montreal, Que.*

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Reprint requests to: Dr. Carlos A. Barba, Department of Surgery, Saint Francis Hospital and Medical Center, 114 Woodland St., Hartford, CT 06105, USA

days, with decreasing abdominal tenderness and a stable hemoglobin level. Gastroscopy was repeated 3 days after admission. It demonstrated blood arising from the ampulla of Vater. The findings shown by the initial gastroscopy were not seen, suggesting the existence of blood clots at that time. Emergency endoscopic retrograde cholangiopancreatography showed multiple filling defects in the intra- and extrahepatic ducts and in the gallbladder, consistent with clots (Fig. 1).

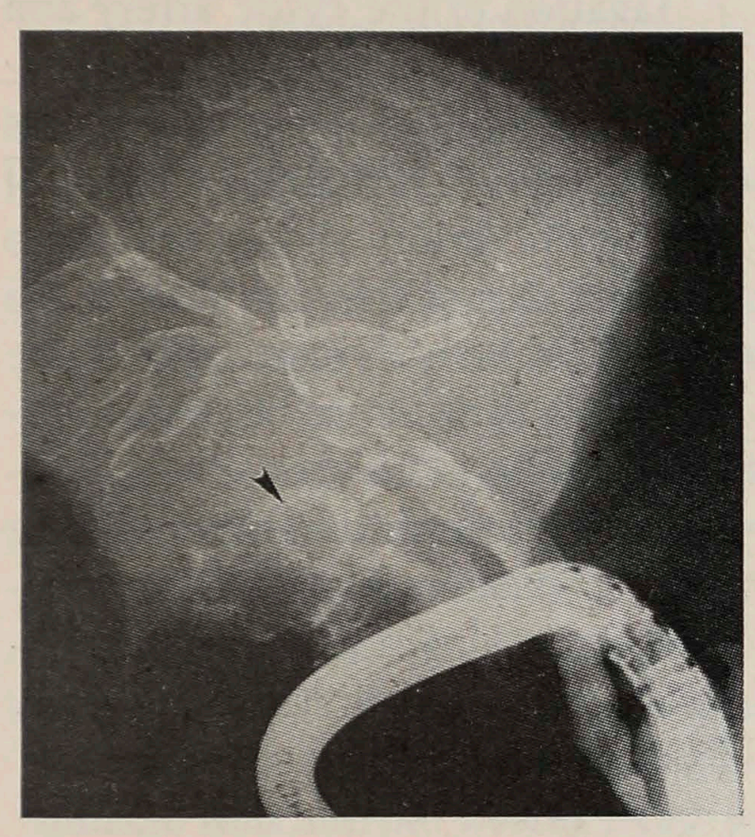

FIG. 1. Endoscopic retrograde cholangiopancreatography showing multiple defects representing blood clots in intra- and extrahepatic bile ducts, as well as in gallbladder. One round filling defect (arrow) in gallbladder has appearance of stone.

Hepatic arteriography showed an aneurysm of a branch of the right hepatic artery. Embolization with coils was attempted, but opacification of the aneurysm persisted (Fig. 2).

The patient was transferred to the surgical intensive care unit for monitoring. Repeat arteriography with further embolization was performed the following day. Although several coils were again placed in the hepatic artery proximal to the aneurysm, it still opacified faintly after the procedure (Fig. 2).

The patient remained hemodynamically stable for the next few days but had persistent right upper quadrant tenderness and a low-grade fever. Colour Doppler ultrasonography was performed on the 8th day after admission to assess patency of the hepatic artery. During the examination, the aneurysm was shown to be in the wall of the gallbladder not in the liver. Both the hepatic artery and the aneurysm had persistent flow (Fig. 3). Review of the angiogram and of the initial ultrasonogram confirmed the diagnosis of aneurysm of the cystic artery.

At operation a gallbladder adherent to duodenum and colon was identified. After the hepatic and gastroduodenal arteries were controlled, the cystic artery was dissected and ligated, and the gallbladder was removed. A saccular aneurysm approximately 2 cm in diameter surrounded the cystic artery. This finding was confirmed on pathological examination: a saccular dilatation of the cystic artery was noted, surrounded by acute and chronic inflammation. This dilatation communicated with the gallbladder wall, which contained areas of focal necrosis.

The patient's postoperative course was uncomplicated and he was discharged from the hospital 1 week after operation. When seen at 2 and 6 weeks after operation, his recovery was good, he had no abdominal pain and tolerated a regular diet.

Discussion

Sandblom[1] was the first to apply the term "hemobilia" to the syndrome of gastrointestinal bleeding caused by hemorrhage into the biliary tract.

The commonest cause of this entity is hepatic trauma, followed by infection and aneurysms of the hepatic or gastroduodenal artery. Hepatic tumours are a rare cause.[2]

A search of the literature revealed only six cases of hemobilia caused by a pseudoaneurysm of the cystic artery.[3–8] All of these cases, like ours,

FIG. 2. (Left) Hepatic angiogram shows aneurysm (arrow) arising from small branch of right hepatic artery. (Middle) After embolization with coils aneurysm still opacifies. (Right) Second embolization procedure was performed with more coils introduced into hepatic artery. However thrombosis of aneurysm (arrow) was not achieved.

were associated with severe cholecystitis and cholelithiasis. We agree with the concept that early thrombosis of the cystic artery, as a reaction to a nearby inflammatory process, may explain the rarity of this entity in spite of the high incidence of acute cholecystitis.[6,7] The difference between a pseudoaneurysm and an aneurysm is that the pseudoaneurysm does not have all the arterial wall layers. In a pseudoaneurysm, fibrotic material and adjacent structures surround an arterial leak or injury. This reaction is usually caused by chronic inflammation reaction.

Selective hepatic arteriography is the diagnostic procedure of choice when hemobilia is suspected. This procedure permits identification of the aneurysm and allows therapeutic management.

In our literature review, the diagnosis was established through angiography in three of the six patients. In two patients the diagnosis was made at laparotomy for uncontrolled upper gastrointestinal bleeding, and in one the diagnosis was made by computed tomography of the upper abdomen. However, because in our patient the cystic artery arose more distally along the right hepatic artery, the angiographic findings were confusing, and a diagnosis of hepatic artery aneurysm was made. The correct diagnosis of cystic artery pseudoaneurysm was made by ultrasonography, which showed the aneurysm as a round, echoic structure arising from the wall of the gallbladder. Colour Doppler imaging showed flow within the aneurysm that could be connected to the hepatic artery through the use of different imaging planes. The colour Doppler imaging showed the aneurysm, its location within the gallbladder wall and persistent flow in it. This is the only reported case of pseudoaneurysm of the cystic artery in which ultrasonography was used to make the diagnosis.

The natural history of an untreated or inadequately embolized aneurysm or pseudoaneurysm is not described in the literature. The major potential complication is severe bleeding. In all the previously reported cases, surgery was required for significant bleeding. We decided to operate on our patient because of the inadequacy of embolization and the persistence of mild abdominal pain, even though the patient was hemodynamically stable and showed no evidence of major gastrointestinal bleeding.

Ligation of the cystic artery and cholecystectomy should be considered the treatment of choice of this rare condition. Proximal control of the hepatic artery should always be the first step of this operation if the patient is hemodynamically stable and there is no active bleeding. Serious bleeding is a high risk when dissection is performed in an area where there is evidence of acute inflammation containing a pseudoaneurysm. In an urgent situation, hepatic artery ligation may be lifesaving.

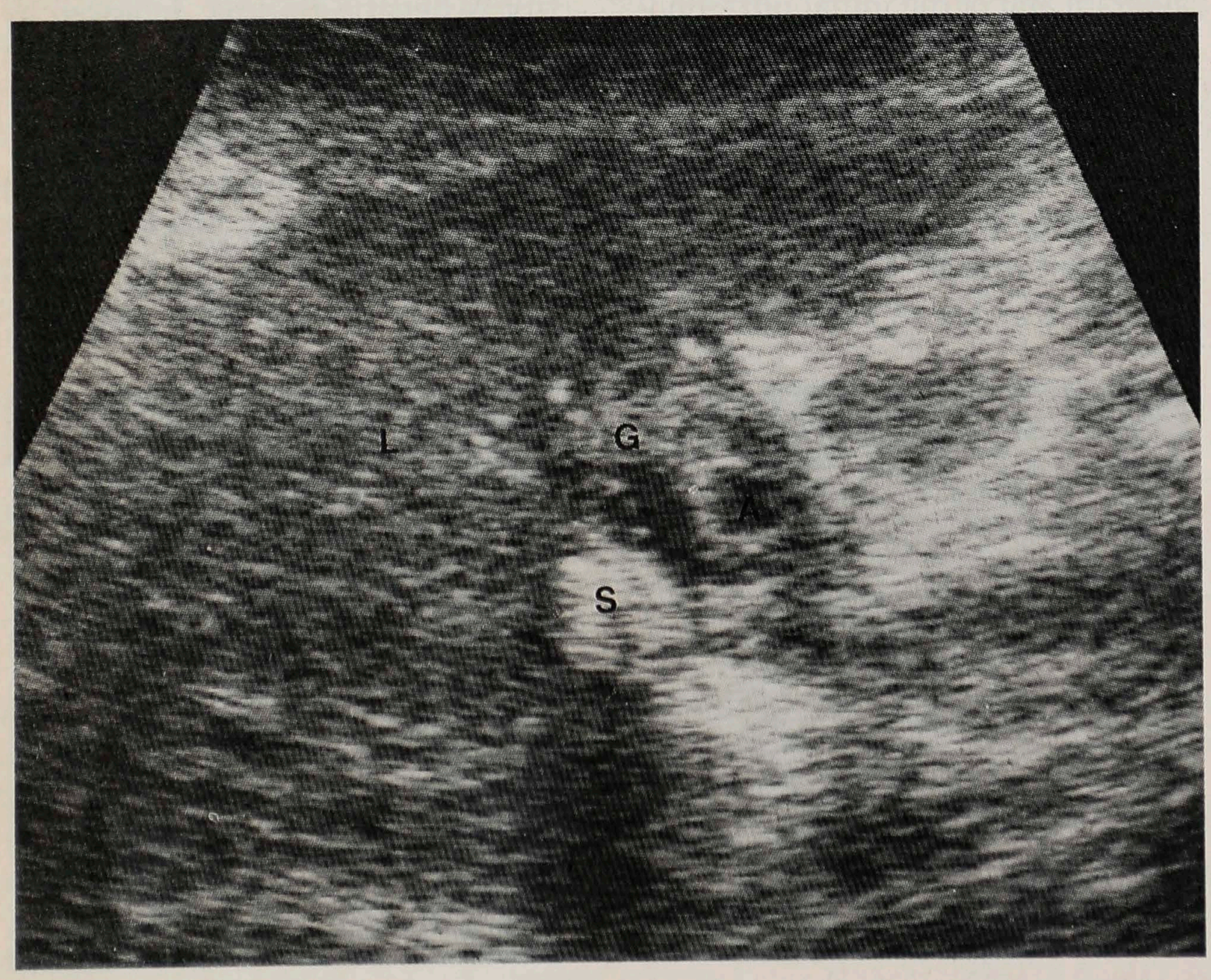

FIG. 3. Colour Doppler ultrasonography. Oblique scan through right upper quadrant shows liver (L) and gallbladder (G). Gallbladder contains stone (S), and aneurysm (A) is visible in gallbladder wall.

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A Variant of Poland's Syndrome

M. Clifford Fabian, MB, ChB; James D. Fischer, MD, FRCSC, FACS

A case of Poland's syndrome — the association of congenital thoracic abnormalities with ipsilateral syndactyly — in a newborn infant is described. The infant demonstrated unusual manifestations of the syndrome: there was extension of the liver through the chest, and there was absence of a whole arm rather than hypoplasia and syndactyly. The authors describe the investigation and treatment of this infant, and they discuss the diversity of findings in Poland's syndrome.

On décrit un cas de syndrome de Poland (l'association d'anomalies thoraciques congénitales et d'une syndactylie ipsilatérale) chez un nouveau-né. L'enfant a présenté des manifestations inhabituelles de ce syndrome : on a noté une extension du foie dans le thorax et il y avait absence totale d'un bras plutôt qu'une hypoplasie ou une syndactylie. Les auteurs décrivent l'investigation et la prise en charge de cette enfant et ils commentent la diversité des observations dans le syndrome de Poland.

In 1841 Alfred Poland, a student demonstrator of anatomy at Guy's Hospital in London, England, described the association of congenital thoracic abnormalities with ipsilateral syndactyly. On dissection of the cadaver of a man who had had difficulty lifting his left hand, Poland found an absent pectoralis minor muscle and sternal costal head of the pectoralis major muscle, hypoplasia of the serratus anterior external oblique muscle and muscles of the left arm as well as syndactyly of the hand.[1] The term Poland's syndactyly was first used to describe this group of congenital anomalies by Clarkson in 1962,[2] although the observation had been made by many others before then. We describe a case of an infant with Poland's syndrome associated with unusual manifestations.

Case Report

At a rural hospital, a 24-year-old primigravida was delivered at 37 weeks' gestation, of a 3500-g, viable female infant with a phocomelia, a chest wall defect and a possible diaphragmatic hernia, all on the left side.

The infant's parents were both healthy adults, with no congenital abnormalities. Both sets of grandparents were of different European extract, so there was no possibility of consanguinity. The father was one of a twin pregnancy, the other child had died shortly after birth of unknown cause. Both parents came from large families in which there was no history of miscarriages, stillbirths or congenital deformities.

The only problems the mother experienced during her pregnancy were mild gestational diabetes, controlled with diet, and a urinary tract infection treated early in the pregnancy. The patient had undergone ultrasonography twice during the pregnancy, but no deformities were detected. There was no history of alcohol abuse, smoking or any other teratogenic drugs taken during the pregnancy. The mother had, however, used acetylsalicylic acid or acet-aminophen occasionally for severe headaches.

The mother had had spontaneous rupture of membranes, and labour had been induced the following day. An assisted vaginal delivery (vacuum extraction) had been carried out, because of fetal bradycardia and maternal exhaustion. The Apgar scores were 8 and 9 after 1 and 5 minutes respectively. The infant was given minimal resuscitation but had a cyanotic spell in the radiology department soon after birth.

On initial examination at our hospital, the infant's weight, height and head circumference were close to the 90th percentile. The infant appeared to be term and was pink in room air. She did not seem to be in any distress and appeared quite content despite her handicaps. There were three small abnormal digits at-

From the Department of Surgery, University of Alberta, Edmonton, Alta.

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Reprint requests to: Dr. James D. Fischer, Division of Pediatric Surgery, 2416–11234 Anderson St., Loma Linda, CA 92354, USA

tached to a small stump arising from the left shoulder (Fig. 1). There was a deformity of the left anterior chest wall extending from approximately the second to sixth intercostal spaces, with a lobular protrusion of soft tissue from the centre of the defect (Fig. 2). Clinically, this appeared to be either pleural or muscular tissue.

Initial investigations demonstrated a normal hematologic and biochemical profile. Chest radiography demonstrated possible dextrocardia, which was confirmed by electrocardiography and two-dimensional echocardiography. There was also a hypoplastic left scapula, left phocomelia, absence of the left chest musculature, a hypoplastic left lung with elevation of the left hemidiaphragm accompanied by herniation or eventration of abdominal contents into the left hemithorax (Fig. 3). Abdominal ultrasonography, upper gastrointestinal series (Fig. 4) and computed tomography of the chest confirmed these findings, but we could not determine exactly what was in the chest and whether or not the diaphragm was intact. The computed tomography scan also showed a large soft-tissue mass of unknown origin in the left anterior side of the chest.

The external defect of the left chest wall was excised under general anesthesia. This seemed to connect to a red, firm tissue mass within the subcutaneous tissue of the chest wall. Histologic examination confirmed this to be liver tissue, including hepatocytes, portal triads, extramedullary hematopoiesis and quite large areas of bile-duct proliferation outside the parenchyma. On exploratory laparotomy, there was a large eventration of the left hemithorax, but the diaphragm was intact. There was extension of the left hepatic lobe into the left chest wall; this lobe had extruded itself through the chest wall defect and presented as an external mass. The remaining portion of the liver and gallbladder looked entirely normal, and the right hemidiaphragm was normal. There was a pancreatic rest on the first position of the jejunum just beyond the ligament of Treitz.

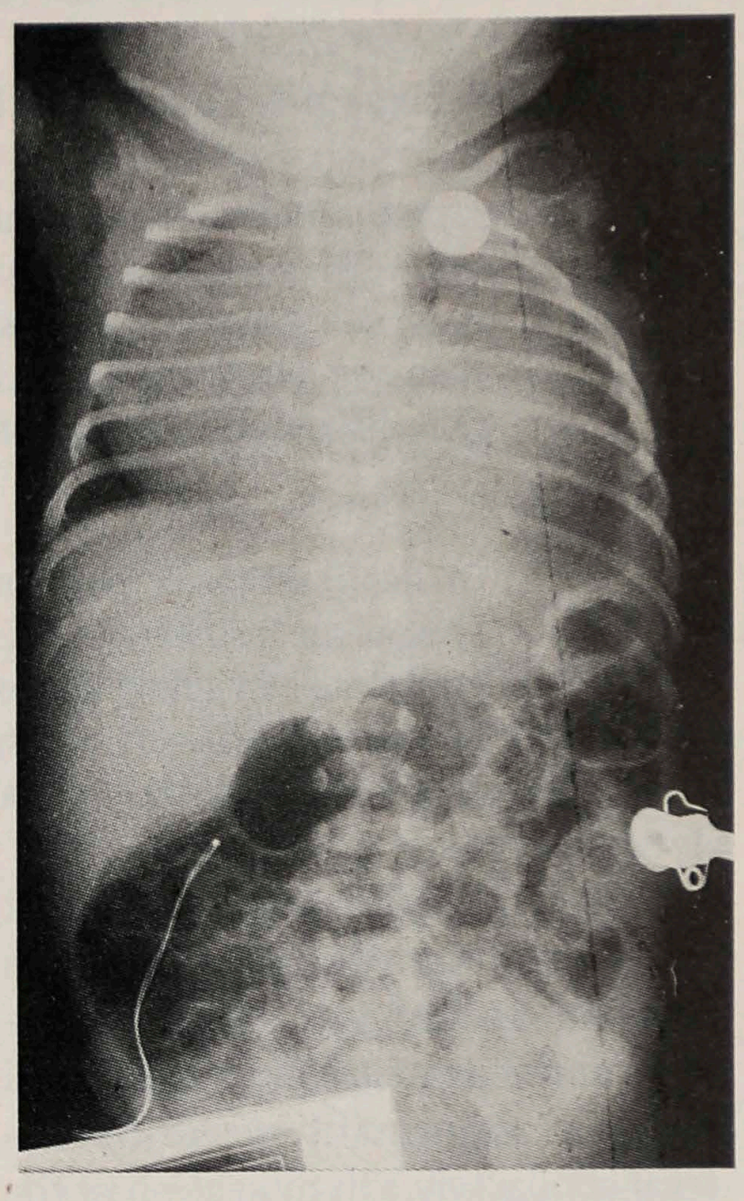

FIG. 3. Chest film demonstrating dextrocardia and hypoplastic left scapula with either eventration or herniation of abdominal contents into left thoracic cavity.

FIG. 1. Chest wall defect with three small, abnormal digits attached to small stump adjoined to left shoulder.

FIG. 2. Close up of lobular protrusion of soft tissue from chest wall defect. Protrusion was found to be made up of liver tissue.

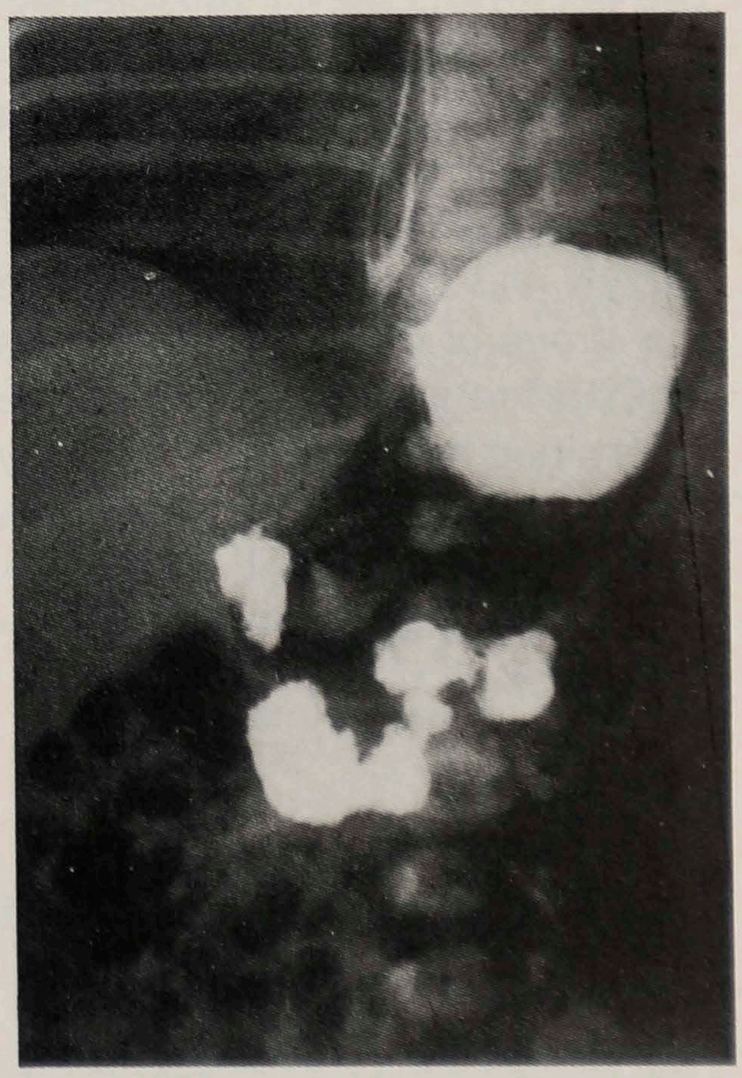

FIG. 4. Gastrointestinal series confirms abdominal contents in left thoracic cavity, most likely involving fundus of stomach.

The appearance of the spleen and kidneys was unremarkable as were the uterus and fallopian tubes, but there were bilateral streak ovaries. No corrective procedure was done at this stage because the infant was asymptomatic.

Two-year follow-up of the infant revealed good growth and development with no respiratory or gastrointestinal compromise. A prosthesis was applied to the left upper limb and no further treatment has yet been planned for the residual chest wall defect.

Discussion

This infant with Poland's syndrome presented with unusual gastrointestinal and chest abnormalities. Extension of liver into the chest with extrusion through the chest wall has not been described previously. The presence of a phocomelia was also unusual, because Poland's syndrome classically includes hypoplasia of the arm muscles and syndactyly rather than the absence of an arm. Perhaps the findings in this case should be considered as a variant of Poland's syndrome. Other articles have emphasized the diversity of this syndrome with many associated problems, including renal agenesis, acute lymphoblastic leukemia, hemivertebrae,[3] scoliosis, cervical ribs, dextrocardia[4] and duplication ipsilateral of the renal collecting system.[5]

More recently, hypotheses have been made of a vascular etiology for Poland's, Klippel–Feil and Möbius' syndromes. It has been proposed that these conditions are the result of an interruption of the early embryonic blood supply of the subclavian arteries, the vertebral arteries and their branches and that the occlusions occur at specific locations in these vessels during or around the 6th week of embryonic development. The term subclavian artery supply disruption sequence is suggested for the group of birth defects represented by the above three conditions.[6]

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Sudden Rupture of an Indirect Inguinal Hernial Sac With Extravasation in Two Patients on Continuous Ambulatory Peritoneal Dialysis

Anthony Ralph-Edwards, MD, FRCSC;* Donna Maziak, MD, CM;* Mervyn Deitel, MD, FRCSC;* Douglas A. Thompson, MD, FRCPC;† Daryl S. Kucey, MD;* T. Arnold Bayley, MD, FRCPC‡

Continuous ambulatory peritoneal dialysis (CAPD) is commonly used to treat patients suffering from end-stage renal failure. This method can be used on a long-term basis and in the short term for patients awaiting renal transplantation. Inguinal hernias are a potential source of morbidity for these patients. Therefore, when patients with inguinal hernias require long-term peritoneal dialysis, the hernia should be repaired if the patient's medical status will allow it. The authors report on two patients maintained on CAPD who experienced rapid enlargement and perforation of a previously undiagnosed asymptomatic inguinal hernia. The hernia was repaired successfully, without complication, in both cases.

La dialyse péritonéale ambulatoire continue (DPAC) est fréquemment utilisée pour traiter les patients en insuffisance rénale terminale. Cette méthode peut être employée à long terme, de même qu'à court terme pour les patients en attente d'une transplantation. Les hernies inguinales peuvent être une source de morbidité chez ces patients. En conséquence, quand des patients souffrant de hernie inguinale nécessitent une dialyse péritonéale prolongée, la hernie doit être réparée lorsque l'état du malade le permet. Les auteurs décrivent les cas de deux patients maintenus sur DPAC qui subirent un élargissement rapide et une perforation d'une hernie inguinale asymptomatique, préalablement non diagnostiquée. La hernie fut réparée avec succès, sans complication, dans les deux cas.

Continuous ambulatory peritoneal dialysis (CAPD) is a simple, cost-effective method of managing patients with end-stage renal failure. It is the most common form of home dialysis. Studies have demonstrated no difference in the death rate between patients receiving CAPD and patients receiving dialysis in hospital, when other complicating factors are taken into account.[1] Catheters are often placed under local anesthesia. Few patients are considered medically unsuitable for CAPD, but those with conditions worsened by increased intraperitoneal pressure (e.g., hernias), are unsuitable candidates unless the complicating condition is corrected. We report an uncommon delayed complication in two men on CAPD who had undiagnosed indirect inguinal hernias.

Case Reports

Case 1

A 37-year-old man with advanced idiopathic renal failure was referred for insertion of a CAPD catheter. On thorough history-taking and physical examination, no physical abnormalities were found. A Tenckhoff cannula was placed, and the patient learned the regimen for CAPD without difficulty. With 2000-mL exchanges four times daily, a serum creatinine level in the range of 600 to 700 mmol/L and a serum urea level in the range of 30 to 35 mmol/L urea were maintained. The patient was feeling well and able to carry out his normal daily activities. He experienced no episodes of peritonitis.

Approximately 200 days after insertion of his catheter, the patient first noted a firm mass in his right groin. The right scrotal sac enlarged rapidly and the swelling became fluctuant. This swelling rapidly extended to the left scrotum, penis, lower ab-

*From the *Department of Surgery, †Department of Nephrology and ‡Department of Endocrinology, University of Toronto, St. Joseph's Health Centre, Toronto, Ont.*

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Reprint requests to: Dr. Mervyn Deitel, St. Joseph's Health Centre, 30 The Queensway, Toronto, Ont. M6R 1B5

domen and upper thighs. On physical examination no hernia was discernible. He noted that the swellings were worse after each dialysis. Initially, the patient was treated with low-volume, increased-frequency dialysis (1000 mL six to eight times daily), but there was no improvement. Cannulography, performed with Omnipaque-300 contrast material, demonstrated free flow of dye into the peritoneal cavity with no evidence of retroperitoneal or groin extravasation; however, only 30 mL of contrast medium was injected by the radiologist. Computed tomography (CT) identified a small mass in the right groin (Fig. 1).

The patient was admitted to hospital for elective hernia repair. Approximately 230 days after insertion of the CAPD catheter, an indirect right inguinal hernial sac, which had perforated at its tip, was excised. Postoperatively the swelling resolved over 72 hours, and there was no recurrence.

Case 2

A 62-year-old man with chronic renal failure secondary to diabetic nephropathy was referred for insertion of a CAPD catheter. On physical examination no hernia was discernible. A Tenckoff catheter was inserted without complication. The patient learned the technique of CAPD. With 1500-mL exchanges four times daily, a serum creatinine level in the range of 600 to 700 mmol/L and a serum urea level in the range of 30 to 35 mmol/L urea were maintained. The patient did well on this regimen for 8 months. Then he was seen in the emergency department with a 1-week history of bilateral scrotal swelling that worsened after each dialysis exchange. The swelling tracked down the penis and both thighs equally. On physical examination no inguinal hernia was found in either groin. Serial scans of the abdomen and groins were obtained by injection of technetium 99m sulfur colloid (7 mCu) through the dialysis catheter (Fig. 2); a CT scan with intravenous contrast was also obtained (Fig. 3). The nuclear scan showed radioactivity concentrated in the right groin and scrotum within 5 minutes, consistent with an inguinal hernia; the CT scan also demonstrated the right inguinal hernial sac.

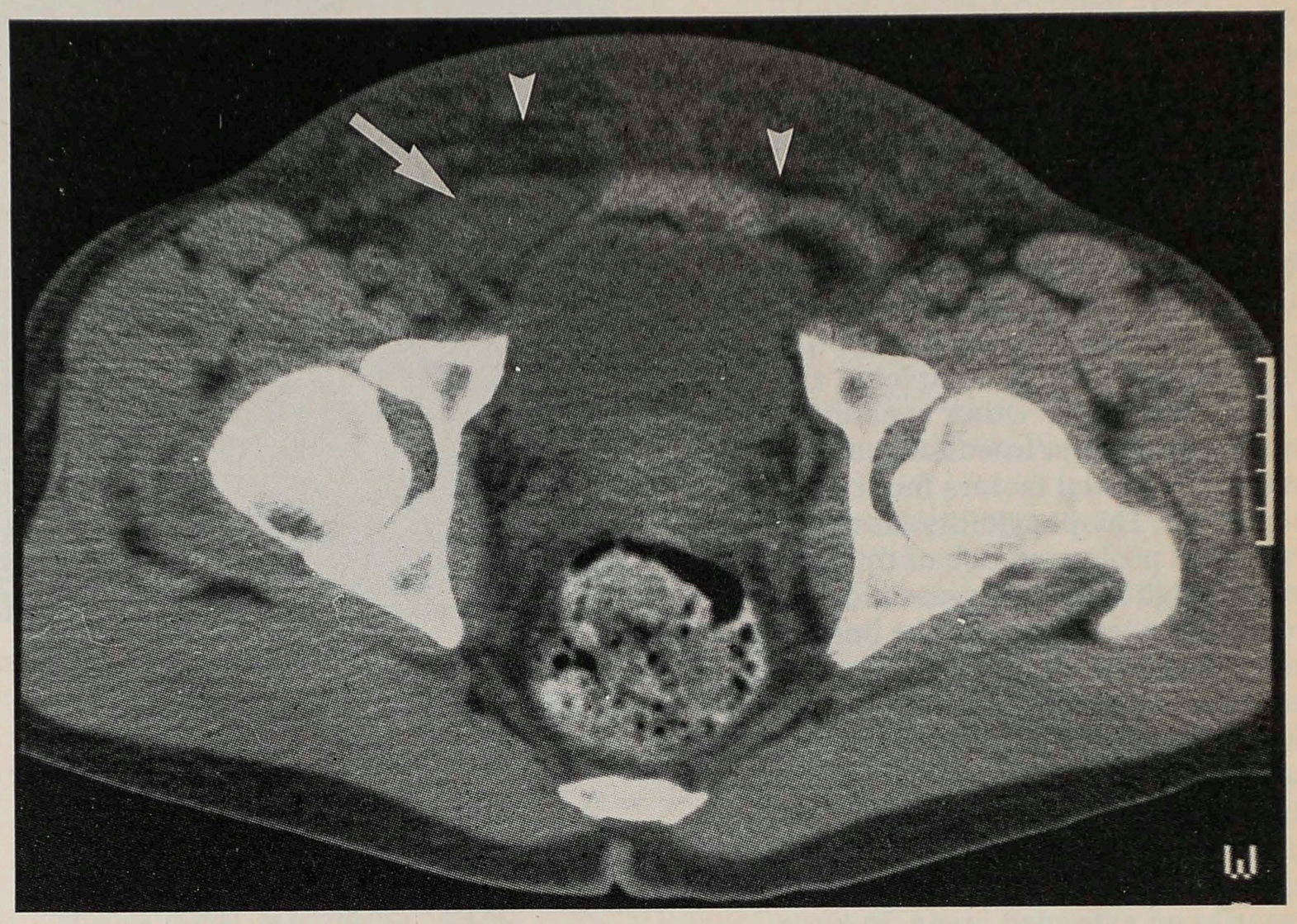

FIG. 1. Case 1. Computed tomography scan identified mass in right inguinal canal (arrow). Edematous subcutaneous adipose tissue can be seen (arrowheads).

FIG. 2. Case 2. Scan of abdomen and groins after instillation of technetium-99m sulfur colloid into dialysate shows radioactivity in right scrotal sac.

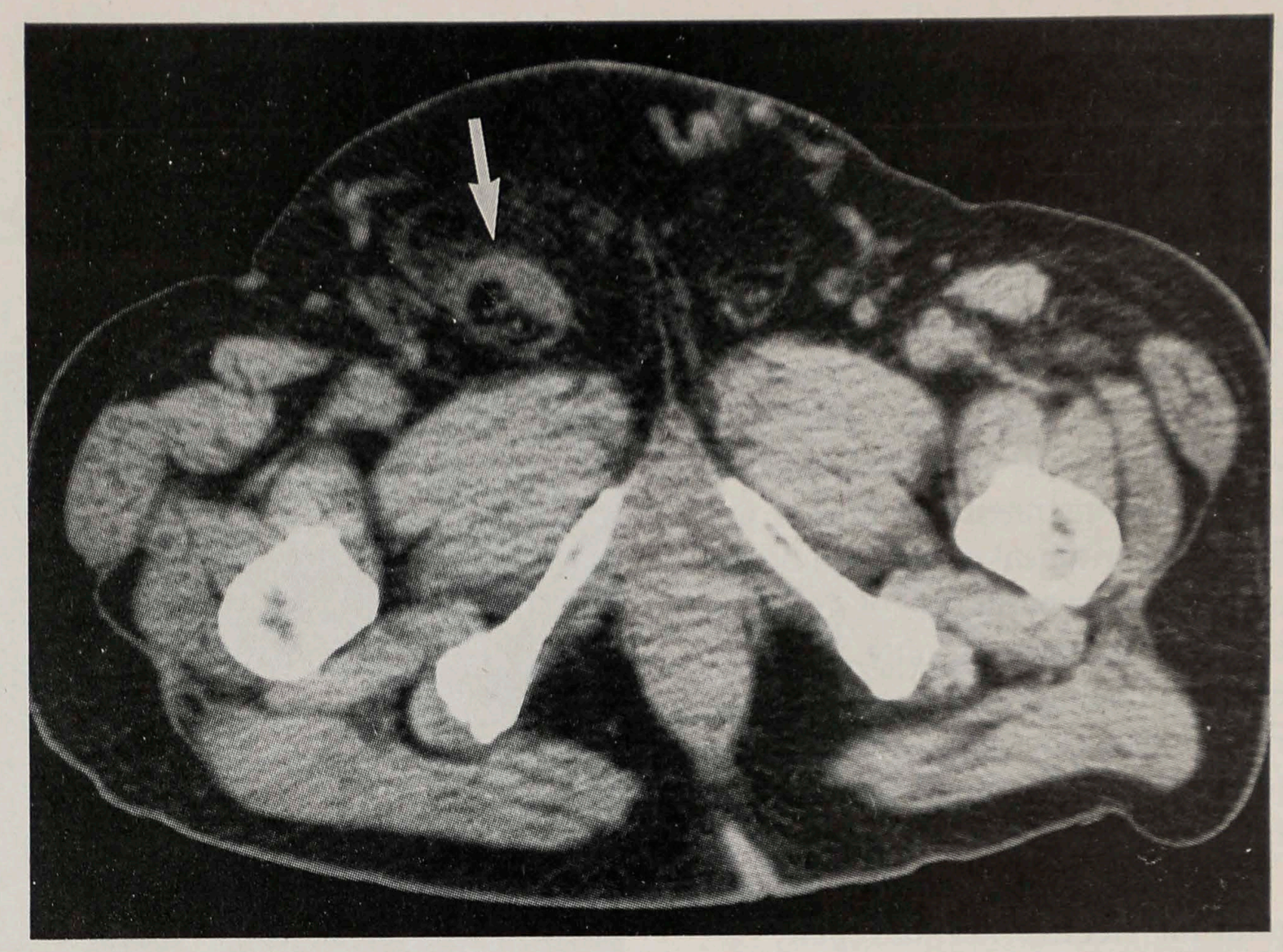

FIG. 3. Case 2. Computed tomography scan indicates mass in right inguinal canal (arrow).

The patient was admitted to hospital for repair of a right indirect inguinal hernia with excision of the perforated hernial sac. The postoperative course was uncomplicated, with rapid resolution of the swelling over 48 hours.

Discussion

Although an inguinal hernia is not an absolute contraindication to placement of a CAPD catheter, it is well known that CAPD, especially with large volumes of dialysate, may gradually enlarge any inguinal hernial sac that is present and exacerbate hernia symptoms.[2] For this reason, known inguinal hernias in patients who require CAPD are repaired whenever the patient's medical status will allow. CAPD is also a recognized cause of inguinal hernias in predisposed patients. In the two cases presented, the patients likely had a patent processus vaginalis or a small asymptomatic indirect inguinal hernia at the time of catheter insertion. With continuing CAPD, the sac eventually ruptured, allowing extravasation of dialysate into the subcutaneous tissues. The complication was indicated by the history of an initial acute unilateral groin swelling in one patient and verified by CT. Radioisotopic labelling of the intraperitoneal fluid in the second patient demonstrated extravasation and provided lateralization of the hernial defect. Simple hernia repair, with excision and ligation of the sac and reinforcement of the posterior inguinal wall, resulted in rapid resolution of discomfort and swelling for both patients. They continued to do well on CAPD.

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BOOK REVIEWS
CRITIQUES DES LIVRES

GENERAL SURGERY BOARD REVIEW. 2nd edition. Edited by Michael S. Gold, Larry A. Scher and Gerald Weinberg. Raven Press Ltd., New York. 1993. $68. ISBN 0-88167-958-5

This soft-cover book is intended as a review for the examinations for certification or recertification by the American Board of Surgery. As such, it also serves as an overview of important knowledge required for the general surgery examinations of the Royal College of Physicians and Surgeons of Canada.

Since 1976, certification by the American Board of Surgery holds only for 10 years, after which recertification must be taken. To prepare for these examinations, a review course has been given annually by the Department of Surgery of the Albert Einstein College of Medicine/Montefiore Medical Center, Bronx, NY. This book is an extension of the syllabus of that course. The text covers general surgery in 32 chapters by 37 authors who are local experts in their areas of interest; all but 2 are from the city of New York.

The book will indicate the areas in which the reader may require additional review. Each chapter contains sample multiple-choice questions (the answers are at the back of the book) and ends with a current list of selected references.

The initial chapters cover the esophagus, the stomach, the duodenum, the small bowel, the colon, the rectum, the anus, the biliary tract, portal hypertension and the pancreas. Peripheral vascular and lymphatic disease, the breast, oncology, gynecology, pediatrics, trauma, transplantation, the pituitary and adrenal glands, thyroid and parathyroid glands, thoracic surgery, cardiac surgery, radiology, melanoma, sarcoma, neurosurgery, anesthesia, orthopedic surgery, plastic surgery and urology are well covered

for the general surgeon. Most sentences contain an important fact with no extraneous material, and each chapter is generally well presented by known authors. The chapter on head and neck tumours by Carl Silver is a gem, and there are three excellent chapters by Steven Blau — on burns, critical care and surgical nutrition. The chapter on critical care is superb. The references indicate what specific areas in textbooks should be consulted for further amplification if the reader has a particular interest. Surgical infection is adequately reviewed.

There are a number of helpful algorithms to guide decisions in management, and a number of important tables (e.g., carcinoma of anus and laparoscopic cholecystectomy).

This text is well done and will have general, day-to-day use for those who want to review a certain subject. Those who trained in the city of New York know that the surgeons are generally an elevated, alert group who seem to interact mainly within their own locale. Also, those of us who have undertaken to write surgical texts know that this is a demanding task. The authors have produced a relevant overview, which is valuable for the intended readership.

Mervyn Deitel, MD, FRCSC, FACS
Professor
Department of Surgery
and Department of Nutritional Sciences
University of Toronto
St. Joseph's Health Centre
Toronto, ON
M6R 1B5

CARDIAC SURGERY, CURRENT ISSUES 1. Edited by Auriel C. Cernaianu and Anthony J. DelRossi. 212 pp. Illust. Plenum Press, New York. 1992. $75 (US). ISBN 0-306-44319-8

This monograph comprises the presentations from the 4th annual symposium entitled "Cardiac Surgery: 1992" and held in the US Virgin Islands.

The title of the book indicates that it is dedicated to current issues in cardiac surgery, written by expert cardiac surgeons, nurses and perfusionists. The information contained in the monograph is readily available in current journals and textbooks. Furthermore, not all current issues in the field are covered. Examples of these omissions include myocardial protection and homograft valves. Cardiac transplantation, cardiac assist devices and the artificial heart are covered only briefly.

The chapters on myocardial assistance with skeletal muscle, extracorporeal membrane oxygenation and complex cardiac problems are excellent.

Overall, this monograph will be of value for those who need information on particular cardiac topics without the detail that a practising cardiac surgeon would require. Medical students, nurses and perfusionists will find the book easy to read and informative.

Tomas A. Salerno, MD, FRCSC, FACS
Professor of surgery
Division of Cardiovascular and Thoracic Surgery
St. Michael's Hospital
30 Bond St.
Toronto, ON
M5B 1W8

TECHNIQUES IN THERAPEUTIC ARTHROSCOPY. Edited by J. Serge Parisien. 385 pp. Illust. Raven Press Ltd., New York. 1993. $125 (US). ISBN 0-7817-0054-X

This book on therapeutic techniques in arthroscopy is divided into six sections on the basis of joint anatomy. All current arthroscopic procedures are covered by recognized authorities. The relative emphasis on the knee and the shoulder is appropriate, considering the volume of surgery performed in these joints. Similarly, the brief mention of the elbow and hip is appropriate.

The book is beautifully illustrated. It contains throughout full colour photographs of arthroscopic views of the joints for the techniques covered. These are associated with and augmented by line drawings. The selection of the illustrations greatly enhances the value of this text.

The shortcoming of this book is that it is purely a technical reference. Results are not critically discussed, and in many

cases the follow-up for the procedures is limited. Therefore many of the techniques described must be considered still experimental. Each chapter contains an up-to-date list of references that includes articles published as recently as 1992. This will be a useful source of information for further reading regarding the outcome of the described procedures.

Overall, this is a superbly produced book that fulfils its objective in providing up-to-date, practical information for techniques in arthroscopic surgery.

All surgical practitioners who use arthroscopic techniques will find this book of value. Those who perform a lot of arthroscopic surgery will benefit from different viewpoints, and those starting out will find it most helpful in the clear demonstration of the technical details. Fellows and residents in training will find the book a useful reference source. This volume should be in the libraries of institutes in which orthopedic training is carried out.

Darrell J. Ogilvie-Harris, MB, FRCSC
Chief of orthopedic surgery and
Smith and Nephew Richards Professor
The Toronto Hospital
Western Division
399 Bathurst St.
Toronto, ON
M5T 2S8

THE ELBOW AND ITS DISORDERS. 2nd edition. Bernard F. Morrey. 903 pp. Illust. W.B. Saunders Company/Harcourt Brace Jovanovich, Inc., Philadelphia. 1993. $216. ISBN 0-7216-6794-5

The second edition of Dr. Morrey's comprehensive text represents a remarkable improvement on an already outstanding textbook. Seventeen chapters have been added. The text is comprehensive in every way and is recognized as "the" textbook on the elbow. It has no less than 62 contributors, and Dr. Morrey has written 25 of the 65 chapters.

This edition seeks to focus on recent developments that have occurred in surgical technique. In particular, surgical innovations of the past decade are explained in greater detail, and some are described for the first time. The new edition is more than just an update of the first edition. It contains a great deal of new information; for example, distraction arthroplasty, semiconstrained arthroplasty, ulnohumeral arthroplasty and the latest techniques of ligamentous reconstruction are discussed in detail.

The book is profusely illustrated. The reference lists are comprehensive. The text is easy to read and remarkably uniform considering the number of contributors. In fact, the text is so exhaustive that I wish Dr. Morrey would write a text about each joint in the body! The great breadth of subjects that are discussed in detail is such that one can learn a great deal about orthopedics in general.

There is no question that this book deserves a place in the library of every institution where orthopedic surgery is practised and in the personal library of every orthopedic surgeon. Orthopedic trainees should refer to this text whenever they are confronted with a problem relating to the elbow. The book is a testament to Dr. Morrey's intellectual energy and should be recognized as such. It was a great pleasure to review this text, which I highly recommend.

Robin R. Richards, MD, FRCSC
Associate professor
Department of Surgery
University of Toronto.
Head
Division of Orthopedic Surgery
St. Michael's Hospital
30 Bond St.
Toronto, ON
M5B 1W8

SURGICAL ANATOMY OF THE TEMPORAL BONE. 4th edition. James A. Donaldson, Larry G. Duckert, Paul M. Lambert and Edwin W. Rubel. 562 pp. Illust. Raven Press, Ltd., New York. 1992. $130 (US). ISBN 0-88167-915-1

Since its introduction in 1967, this book, initially edited by Drs. B.J. Anson and J.A. Donaldson, has been considered the definitive text on the subject. Subsequent editions have reflected the expansion of the field of otology. This is the first edition that has appeared without the contribution of Dr. Anson, a highly respected anatomist who died in 1974. Three new editors have assisted Dr. Donaldson in the production of this edition.

Dr. Rubel has revised the section on the developmental anatomy of the ear. This chapter concisely describes the embryology of the ear along with its clinical significance.

In recent years considerable advances have been made in neurotology and lateral skull-base surgery. Dr. Lambert has done excellent work in expanding the section on adult anatomy to reflect these developments in otology.

A brief guide to temporal bone dissection completes the book.

As always the greatest strength of this work is the numerous anatomic diagrams. These detailed illustrations allow the reader to achieve an excellent understanding of the intricacies and variations of temporal bone anatomy.

I have two criticisms of this book. First, because the illustrations are placed together at the end of each section, it can be difficult to follow the narrative. Second, the guide to temporal bone dissection is limited; the reader should turn to one of the several other excellent dissection manuals to become familiar with the surgical anatomy of the temporal bone through dissection.

This latest edition of the *Surgical Anatomy of the Temporal Bone* provides an excellent description of the embryology and surgical anatomy of the temporal bone. For the otolaryngologist who specializes in otology, neurotology and lateral skull-base surgery, this book is highly recommended.

David Schramm, MD, FRCSC
Clinical lecturer
Department of Otolaryngology
University of Ottawa.
Suite 521
267 O'Connor St.
Ottawa, ON
K2P 1V3

Reviewers 1993/Examinateurs 1993

The Coeditors, on behalf of the Editorial Board of the *Canadian Journal of Surgery* acknowledge with thanks the following reviewers of manuscripts for the past year.

Au nom du conseil de rédaction du *Journal canadien de chirurgie*, les corédacteurs désirent remercier les personnes suivantes qui ont examiné des manuscrits au cours de l'année écoulée.

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The *Canadian Journal of Surgery* will consider manuscripts of original articles, clinical research, surgical technique, clinical reviews, history of surgery, a limited number of case reports, editorials and letters. Four copies of manuscripts, in English or French, should be submitted to Dr. Roger G. Keith, *Canadian Journal of Surgery,* Department of Surgery, Royal University Hospital, 103 Hospital Dr., Saskatoon, SK S7N 4J9.

A covering letter, signed by all the authors, should state that the manuscript has not been published previously and is not under consideration by any other journal. The authors should include a signed letter of permission from people identified in the acknowledgements or identifiable in illustrative material as well as from the copyright holder of previously published material (e.g., tables, illustrations and long quotations) that is being reproduced, with or without modification, in the submitted article. The authors must disclose the source of any financial or material support, any commercial interest they may have in the subject of the study and any affiliation or involvement with an organization that has a financial interest in the research materials used or the topic.

Manuscript Preparation

The style of the submission should be compatible with "Uniform requirements for manuscripts submitted to biomedical journals" (*Can Med Assoc J* 1994; 150: 147–154).

The authors' names should appear on the title page and the back of each set of illustrations. All acknowledgements should be placed on a separate unnumbered page after the list of references.

To facilitate editing and electronic scanning, all pages (title page, abstract and key words [MeSH terms if possible], text, references and figure legends) must be double-spaced, in 10-cpi, letter-quality type, without right justification or proportional spacing.

For all manuscripts, authors should submit an original and three high-quality photocopies or additional printouts of the text and tables, and four camera-ready copies of the figures.

In writing a case report it is not necessary to give a detailed patient history and results of physical examination in the standard clinical format. Negative findings and normal results of laboratory tests need be included only if they are essential for ruling out a possible diagnosis. It is enough to establish the reasons for the diagnosis and the management. The clinical course should be described briefly and the significant observation or event described in sufficient detail to establish its credibility. Reference to the literature should be confined to supporting the principal point being made about the event or observation.

Abstracts are required for original, review and history articles and for case reports, but not for articles on surgical technique and editorials. A structured abstract should be provided for original and review articles (see October 1992 issue, pages 473 to 475). Abstracts for history articles and case reports should be brief, but detailed (from 60 to 150 words long).

References should be cited in numerical order of their appearance in the text. References cited in tables should be numbered according to where the table is first cited in the text. The style for references should be that used in this issue of the Journal.

Other Considerations

Colour figures can be reproduced only at the author's expense. Authors should submit a positive transparency and three colour prints of each figure.

If the manuscript has been prepared on a computer-based word-processing system, authors must specify the software program used. We edit manuscripts with an IBM-compatible word-processing system and cannot edit from diskettes that are not IBM-compatible. Authors should indicate if they are willing to send us a diskette when their manuscript has been accepted.

The process of initial consideration, peer review and editorial decision making of the manuscript usually takes about 8 weeks. The original copy of a rejected manuscript will be returned to the authors; all other copies will be destroyed.

Accepted manuscripts will be edited not only to conform with *Canadian Journal of Surgery* style and for correctness of grammar, syntax and punctuation but also for clarity. The corresponding author will receive a copy of the edited manuscript *or* a galley proof before publication and is responsible for obtaining coauthors' approval of the changes.

Authors will be expected to sign a document transferring copyright to the *Canadian Journal of Surgery*. All accepted manuscripts become the permanent property of the Canadian Medical Association and may not be published elsewhere, in whole or in part, without written permission of the publisher. Reprints will be available for purchase and may be distributed as the author desires.■

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Le *Journal canadien de chirurgie* étudiera les manuscrits d'articles originaux, de recherche clinique, de technique chirurgicale ou d'histoire de la chirurgie, un nombre restreint d'études de cas, des éditoriaux et des lettres. Quatre exemplaires des manuscrits, en français ou en anglais, doivent être adressés au D[r] Roger G. Keith, *Journal canadien de chirurgie*, Département de chirurgie, Royal University Hospital, 103 Hospital Dr., Saskatoon, SK S7N 4J9.

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Préparation du manuscrit

Le style doit se conformer aux <<Exigences uniformes pour les manuscrits présentés aux revues biomédicales>> (*Can Med Assoc J* 1994; 150: 159-167).

Les noms des auteurs doivent paraître sur la page titre, de même qu'à l'endos de chaque jeu d'illustrations. Les remerciements doivent figurer sur un feuillet séparé, non paginé, à la suite de la bibliographie.

Toutes les pages (page titre, résumé et mots clefs [les termes MeSH si possible], textes, références, tableaux et légendes de figures) doivent être dactylographiées à double interligne, à 10 caractères au pouce, sans justification à droite ni espacement proportionnel, et être imprimées en <<qualité lettre>> afin de faciliter la révision et la lecture électronique.

Pour tout manuscrit, les auteurs doivent soumettre un original et trois photocopies de bonne qualité, ou trois impressions supplémentaires du texte et des tableaux, et quatre copies des figures prêtes à la reproduction photographique.

Pour les études de cas, il n'est pas nécessaire de rapporter une anamnèse détaillée avec les résultats de l'examen physique dans le format clinique normal. Les résultats négatifs ou normaux d'examens de laboratoire de doivent être mentionnés que s'ils sont essentiels à l'exclusion d'un diagnostic possible. Il suffit d'établir les raisons qui ont contribué au diagnostic ou au traitement. L'évolution clinique doit être décrite succinctement et les observations ou événements importants doivent être rapportés avec suffisamment de détails pour en établir la crédibilité. La bibliographie doit se limiter au soutien du point que l'on veut faire ressortir quant à l'événement ou à l'observation.

Des résumés sont exigés pour les articles originaux, historiques ou de revue ainsi que pour les études de cas; ils ne sont pas requis pour les articles sur les techniques chirurgicales ou pour les éditoriaux. Un résumé structuré doit être fourni pour les articles originaux et de revue (voir le numéro d'octobre 1992, pages 473 à 475). Les résumés pour les articles historiques et les études de cas doivent être brefs, mais détaillés (de 60 à 150 mots).

La bibliographie doit être établie numériquement, selon d'ordre d'apparition dans le texte. Les références citées dans les tableaux doivent être numérotées d'après l'ordre de la première mention du tableau dans le texte. Le style adopté pour la bibliographie est celui qui est utilisé dans ce numéro du Journal.

Autres considérations

Les figures en couleur sont à la charge de l'auteur. Il lui faudra fournir une diapositive et trois épreuves en couleur pour chaque figure.

Les auteurs sont priés de nous préciser, le cas échéant, le logiciel ayant servi à préparer le manuscrit. Nous révisons les manuscrits avec un système de traitement de texte compatible avec le système IBM. Nous ne pouvons pas réviser à partir de disquettes qui ne sont pas compatibles avec le système IBM. Au moment de la soumission du manuscrit, les auteurs doivent nous indiquer s'ils sont disposés à nous faire parvenir une disquette quand leur texte est accepté.

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Director of Human Resources
Cariboo Memorial Hospital
517 North 6th Ave.
Williams Lake, BC
V2G 2G8
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Dr. V. Taraska, Chairman
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The Winnipeg Clinic
425 St. Mary Ave.
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This 1-year postgraduate fellowship offers a wide range of clinical experience in endourology including percutaneous surgery, ureteroscopy, ESWL and laparoscopic surgery in urology. Approximately 50% of the fellow's time will be spent in the laboratory participating in projects related to endourologic and shock wave lithotripsy research. Salary will commensurate with the level of training.

Please reply with curriculum vitae to:

Dr. John Denstedt
Chief of Urology
St. Joseph's Health Centre
268 Grosvenor St.
London, ON N6A 4V2 –S93-163

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Interested applicants should submit resumes in confidence to:

Dr. M. Terwiel
Medical Director
Ridge Meadows Hospital
and Health Care Centre
11666 Laity Street
Maple Ridge, BC
V2X 7G5
Tel (604) 463-4111, local 233
Fax (604) 463-9939

—S94-174

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SCARBOROUGH GENERAL HOSPITAL

The Division of General Surgery at Scarborough General Hospital, Scarborough, Ontario is currently considering applicants for a recently vacated position.

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Interested applicants should direct enquiries to:

Dr. D. R. Hodgkiss
Chief, Division of General Surgery
3030 Lawrence Avenue East
Suite 415
Scarborough, Ontario
M1P 2T7
Tel (416) 439-4873

—S94-177

NOTICES
AVIS

Trauma and Critical Care — 1994

The American College of Surgeons Western States Committees on Trauma are sponsoring a course entitled "Trauma and Critical Care — 1994" to be held at Caesars Palace, Las Vegas, from Mar. 14 to 16, 1994. The course meets the criteria for 21 hours of credit in Category 1. The course chairman is Dr. K.L. Mattox. For further information contact: Trauma Department, American College of Surgeons, 55 East Erie St., Chicago, IL 60611, USA; phone: (312) 664-4050, ext. 342.

Pediatric Orthopedic Review Course

The Hôpital Ste-Justine in collaboration with the Montreal Children's Hospital and the Shriners Hospital will present the 7th Ste-Justine Paediatric Orthopaedic Review Course (SPORC) from Apr. 6 to 8, 1994, at the Meridien Hotel in Montreal. The course will include sessions on trauma, tumours, infections, the spine, the foot and the hip, a general session and a session on neuromuscular problems. All lectures will be presented in English. For further information contact: Dr. Benoît Morin, Chairman and secretary, SPORC 94, 3175 Côte Ste-Catherine, Montréal, QC H3T 1C5; phone: (514) 345-4876; fax: (514) 345-4755.

Controversies in Breast Cancer

The Faculty of Medicine, University of Toronto, will hold a meeting entitled "Controversies in the Etiology, Detection and Treatment of Breast Cancer: 1994" at The Old Mill, Toronto, on Apr. 7 and 8, 1994. Further information may be obtained from: Continuing Education, Faculty of Medicine, University of Toronto, Medical Sciences Building, Toronto, ON M5S 1A8; phone: (416) 978-2719; fax: (416) 971-2200.

Laser Medicine and Surgery

The American Society for Laser Medicine and Surgery, Inc., will hold its 14th annual meeting from Apr. 8 to 10, 1994, at the Westin Harbour Castle Hotel, Toronto. For further information contact: Dianne Dalsky, Meeting coordinator, American Society for Laser Medicine and Surgery, Inc., 2404 Stewart Sq., Wausau, WI 54401, USA; phone: (715) 845-9283; fax: (715) 848-2493.

International Symposium on Implant Surgery

The Grand Rapids 22nd Annual International Symposium on Implant Surgery for the Hand, Upper Extremity, and Foot will be held Apr. 14 to 16, 1994, at the Blodgett Memorial Medical Center, Grand Rapids, Mich. The symposium will include live surgical demonstrations on closed-circuit television. For further information contact: Dr. Alfred B. Swanson, Blodgett Professional Building, 1900 Wealthy, SE, Suite 290, Grand Rapids, MI 49506, USA.

Society of American Gastrointestinal Endoscopic Surgeons

The 1994 scientific session and postgraduate course entitled "Complications of Laparoscopy & Flexible Endoscopy" will be presented by the Society of American Gastrointestinal Endoscopic Surgeons (SAGES) from Apr. 16 to 19, 1994, at the Opryland Hotel, Nashville, Tenn. For more information contact: SAGES, 11701 Texas Ave., Suite 101, Los Angeles, CA 90025, USA; phone: (310) 479-3249; fax: (310) 479-9744.

Care of the Trauma Patient

The Sunnybrook Health Science Centre is holding a 2-day conference entitled "The Critical Path: a Multiprofessional Approach to the Care of the Trauma Patient" on Apr. 29 and 30, 1994, at the Four Seasons, Inn-on-the-Park Hotel, Toronto. For information contact: Ms. C. Stolarchuk, Conference coordinator, Sunnybrook Health Science Centre, 2075 Bayview Ave., North York, ON M4N 3M5; phone (416) 480-6100, ext. 5904.

Congress on Endoscopic Surgery

The 2nd Asian Pacific Congress of Endoscopic Surgery will be held at the Hong Kong Convention and Exhibition Centre from June 19 to 23, 1995. For information contact: Dr. Sydney Chung, Department of Surgery, The Chinese University of Hong Kong, Prince of Wales Hospital, Shatin, Hong Kong; phone: (852) 636-2627; fax: (852) 645-3602.

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